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X-RAY PELVIMETRY FOR GENERAL USE*

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IT IS a well-established fact that external measurements of the female pelvis, with the exception of the outlet, are not only inaccurate but perhaps seriously misleading. Consequently there has been a distinct trend toward the use of x-ray examination in pelvimetry. The universal acceptance of this valuable means of appraising the obstetric pelvis has been delayed by alleged technical difficulties and expense.

Experience at the Boston City Hospital during the last three years has convinced us that our technic, which is based on the method originated by Ball,[1] of Columbia University, is simple, inexpensive, informative and practicable for general use. This technic requires no expensive equipment, a simple anteroposterior and a true lateral film being sufficient. The various diameters are measured directly from the films, and the target-to-film distance and the object-to-film distance are used in correcting for magnification. The mathematics involved is completed by means of a special calculator§[2] or slide rule,‖[3] both of which are efficient and easy to manipulate, or by the formula, $O = I\frac{D-d}{D}$, in which O is the diameter of the object, I the magnified image as measured on the film, D the target-film distance and d the object-film distance. The fact that the films are taken by the regular technicians creates no disturbance of the x-ray department's busy routine. The measuring is done by the radiologist, and the interpretation is made by the obstetrician, with the films on display in the delivery room at the time of accouchement.

In the beginning we followed the original suggestion of Ball, by which the volume of the fetal head was compared with the volume capacity of the smallest diameters of the inlet and the mid-pelvis. Latterly, however, we have preferred to attempt to visualize the birth canal as a whole, with all its various important diameters and contours and their conformity to the size, direction and shape of the fetal head that is offered for delivery. Thus we determine the contour of the pelvic inlet and classify the type of pelvis; the anteroposterior diameter of the inlet; the anterior and posterior sagittal diameters of the inlet; the transverse diameter of the inlet; the volume capacity of the smallest diameter of the inlet; the bispinous diameter of the mid-pelvis; the volume capacity of the narrowest diameter of the mid-pelvis; the anterior and posterior sagittal diameter of the mid-pelvis; the total anteroposterior length of the mid-pelvis; the width and contour of the greater sciatic notch; the perpendicular length of the fore-pelvis; the contour, length and angulation of the sacrum; the angulation of the coccyx; the relation of the sacrococcygeal platform to the plane of the mid-pelvis; the position of the baby in utero; the degree of flexion of the fetal head; the amount of molding if labor is in progress; the extent of engagement of the fetal head; the part of the pelvis into which the head is being guided by the cervix; the occipitofrontal diameter of the fetal head if it presents in the transverse diameter of the pelvis; the volume of the fetal head, as derived from the mean circumference; the weight of the baby, as roughly determined by the size of the head; and the presence of twins or fetal monstrosities and bony anomalies of the pelvis.

In order to ascertain the accuracy of the pelvic measurements by this technic, we x-rayed 50 patients, employing both the Thoms[4] and Ball methods. Using the anteroposterior and lateral

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§The Ball Calculator, obtainable from the General Electric X-Ray Corporation, Chicago.

‖The Pelvimetry Slide Rule, obtainable from the Picker X-Ray Corporation, New York City.

diameters of the inlet as criteria, there was no greater variation than 0.5 cm. between the two technics. The accuracy of the Thoms method has been proved at operation by Thoms himself and also by Schuman,[5] who in 1936 reported 50 cases measured at laparotomy; the Thoms technic was accurate within 2 mm. or less.

The study included in this report concerned the routine measuring of 200 unselected primiparas and a correlation of the findings.

The x-ray measurements of the fetal heads were compared with the actual dimensions obtained at the time of birth and proved to be of definite practical value. By assuming a gain in mean circumference of 0.32 cm. and in occipitofrontal diameter of 0.25 cm. weekly during the last month,—according to the law of uniform fetal growth ratios as established by Scammon and Calkins,[6]—one can anticipate fairly accurately the size of the fetal head at the time of labor, even when the films have been taken three or four weeks before delivery. In cases where the head was engaged when the films were taken, the measurements, as a rule, varied less than 0.5 cm. from the actual dimensions. If the head was floating there was an occasional discrepancy up to 1.0 cm. in the mean circumference or 0.5 cm. in the occipitofrontal diameter, but in the large majority of cases the head measurements were sufficiently accurate for making a practical appraisal of the size of the presenting part. In cases of breech presentations the head measurements were of no value.

The contour of the inlet, which is delineated so clearly by the Thoms technic, is not shown so well by the simple anteroposterior film with the patient flat on the table; the inlet is at an angle rather than directly parallel to the film. However, since enough of the fore-pelvis can be seen to gain a general idea of the type-tendency, one can diagnose the type quite correctly by supplementing the anteroposterior, lateral, anterior and posterior sagittal diameters of the inlet. A folded sheet under the lumbar region when the anteroposterior film is taken helps considerably in demonstrating the contour of the inlet. In borderline cases one may add to the anteroposterior and lateral films a third view, that with the patient semirecumbent in the manner of the Thoms technic, in order to visualize more accurately the inlet configuration.

Technic

The technic of measurement is as follows:

Take anteroposterior and lateral films, using a standard target-film distance (we use the 75-cm. or 30-in. distance described by Ball). Place films (14 by 17 in.) transversely for both views. In the anteroposterior position, a folded sheet is placed under the small of the back, and in the lateral position under the side of the chest, to minimize the distortion of the image of the contour of the inlet. In the lateral position, the thighs are extended so that the femurs do not overshadow the symphysis. Shadows of the ischial tuberosities are included on this film. Sketch in the ischial spines and ischial tuberosities, and then mark points between each of these pairs of bony landmarks. Sketch in the inner border of symphysis.

The various diameters and corrections are as follows:

Anteroposterior Diameter of Inlet. From the promontory of the sacrum to the superoposterior limit of the symphysis.

Transverse Diameter of Inlet. The widest transverse measurement of the inlet on the anteroposterior film.

Anterior Sagittal Diameter of Inlet. From the posterosuperior surface of the symphysis to the point of bisection of the anteroposterior diameter by the transverse diameter.

Posterior Sagittal Diameter of Inlet. From the point of bisection of the anteroposterior diameter by the transverse diameter to the promontory of sacrum.

Anterior Sagittal Diameter of Mid-Pelvis. From the posteroinferior border of the symphysis to the interspinous point.

Posterior Sagittal Diameter of Mid-Pelvis. The continuation of the anterior sagittal line from the interspinous point to where it meets either the sacrum or coccyx.

Bispinous Diameter of Mid-Pelvis. A line drawn between the medial margins of the shadows of the ischial spines on the anteroposterior film.

Pubotuberous Diameter. A line drawn at right angles from the anteroposterior diameter of the inlet to the intertuberous point.

Correction Factor for Pelvic Measurements in Mid-Sagittal Plane. (Anteroposterior diameter of inlet, anterior and posterior sagittal diameters of inlet and mid-pelvis, and pubotuberous diameter.) The distance from the symphysis to 1.2 cm. lateral to the medial limit of the greater trochanter of the femur.

Correction Factor for Transverse Diameter of Inlet. The distance from the spinous process of the first sacral segment to a point on the anteroposterior diameter of the inlet where it is bisected by a line parallel to the height of the sacrum and drawn from the interspinous point.

Correction Factor for Bispinous Diameter. The distance from the interspinous point to a point 1.2 cm. posterior to the sacrum or coccyx on the same plane with the interspinous point.

Correction Factor for Circumference of Head in Lateral Film. The distance from the center of the calvarium in the anteroposterior view to meet a line drawn from the wing of the ilium to the described point on the greater trochanter of the femur.

Correction Factor for Occipitofrontal Diameter of Head and Circumference of Head in Anteroposterior Film. The distance from the center of the calvarium in the lateral film to the spinous process of the first sacral segment.

To the various correction factors derived from the films one then adds the tabletop-film distance. The special calculator, the slide rule or the formula is then brought into play for the necessary mathematics required in reducing the magnified

images, as seen on the films, to the actual dimensions.

After the actual mean circumference is ascertained from the films, 2 cm. must be added to the fetal head measurements to allow for thickness of the scalp.

The volume of the fetal head from its mean circumference and the volume capacity of the smallest diameters of the inlet and mid-pelvis — the anteroposterior and bispinous diameters respectively — may be reckoned from the volume charts that accompany the calculator or slide rule.

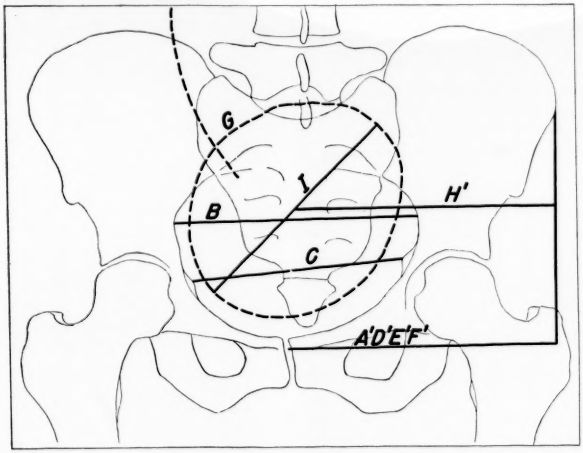

FIGURE 1.

B = transverse diameter of the inlet; C = bispinous diameter; G = circumference of the head, in anteroposterior view; I = occipitofrontal diameter of the head. The primed letters represent their corresponding letter correction measurements; H' = from the center of the calvarium to a line drawn from the image of the iliac wing to a point on the greater trochanter of the femur; A'D'E'F' = from the symphysis to a point ½ inch lateral to the medial limit of the greater trochanter of the femur.

RESULTS

We have adopted a classification of pelves that combines those of Thoms and of Caldwell and Moloy,[7] and is based on both measurements and pelvic configuration. The various types are:

Gynecoid or Female Pelvis. The transverse diameter of the inlet is more than 1 cm. larger than the anteroposterior diameter. The fore-pelvis and posterior pelvis are well rounded. The ischial spines are blunt. The sacrum points backward, affording a wide, greater sciatic notch, and its concavity is well marked. The symphysis is broad, and the pubic arch wide.

The Round Pelvis. This is really a subdivision of the gynecoid, agreeing with it in all details, except that the anteroposterior and transverse diameters of the inlet approximate each other by 1 cm. or less.

The Android or Male Pelvis. The contour of the inlet, rather than the measurements, is important in determining this type of pelvis. The fore-pelvis is acutely angulated. The posterior pelvis is flattened with the greatest transverse diameter of the inlet approaching the sacrum by 3 cm. or less. The spines of the ischium are sharp and abnormally close together. The symphysis is narrow, and the subpubic angle acute. The sacrum is broad and flat and points forward, causing a narrowing of the sciatic notch. The pelvis tends to be funnel-shaped.

The Anthropoid Pelvis. The anteroposterior diameter is greater than the transverse. The pelvis assumes a transversely contracted appearance. The sacrum is long and bayonet-shaped and usually extends straight downward. In this type, also, funnel-shaped pelves are common. Cases of high-assimilation and low-assimilation pelves are fairly frequent.

Platypelloid or Flat Pelvis. The transverse diameter is at least 3 cm. greater than the anteroposterior diameter. In this type the rest of the pelvis may be quite adequate, especially when the sacrum points well backward, allowing a roomy sciatic notch and much wider anteroposterior diameters in the mid-pelvis and outlet than that at the inlet.

Asymmetrical Pelvis. There is a shortening of either oblique diameter, due usually to some accompanying pathologic process in the bony pelvis.

We found but few pure types of the android, anthropoid and flat pelves, but the pure gynecoid

and round types were very frequent. Many mixed types were found, particularly pelves with an android tendency in either the fore-pelvis or the posterior pelvis. The classification of our series is shown in Table 1.

TABLE 1. *Types of Pelves.*

TYPE OR TENDENCY	INCIDENCE %
Gynecoid	65.0
Round	20.0
Android	7.0
Flat	4.5
Anthropoid	3.0
Asymmetrical	0.5

The incidence of the various types of pelves, according to our statistics, differs from those of Thoms and of Caldwell and Moloy mainly because with their method of visualization they were able to note the less obvious differences in contour that we believe are more important from an anthropological than an obstetric viewpoint.

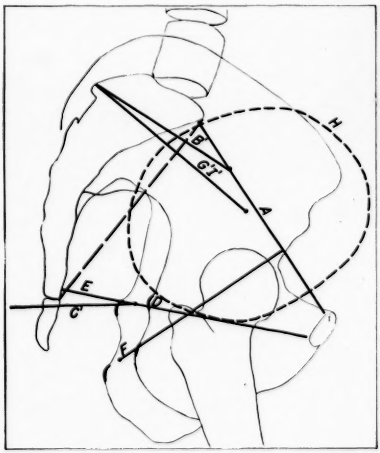

FIGURE 2.

A = anteroposterior diameter of the inlet; D = anterior sagittal diameter of the mid-pelvis; E = posterior sagittal diameter of the mid-pelvis; F = pubotuberous diameter or length of fore-pelvis; H = circumference of the head in lateral view; B' = from the point of bisection of the anteroposterior and transverse diameters of the inlet (this point is obtained by a line drawn parallel to that representing the height of the sacrum and from the interspinous point) to the spinous process of first sacral segment; G'T' = from the center of the calvarium to the spinous process of first sacral segment; C' = from the interspinous point to a point ½ inch posterior to the sacrum or coccyx on the same plane with the interspinous point.

External measurements, which were done routinely on all cases, proved of but slight value in diagnosing the type of pelvis or in ascertaining the correct anteroposterior diameter of the inlet. However, external palpation and mensuration of the subpubic arch and outlet were of unquestionable value. In only 20 per cent of our cases was the conjugate vera, as determined from the measurement of the external conjugate, within 0.5 cm. of the measurement by x-ray, and in some cases there was a discrepancy of 4.0 cm. or more. In no case was the conjugate vera, as measured by x-ray, smaller than the measurement computed from the external conjugate. As a corollary to this latter finding we can assume that if the external conjugate is ample, which usually means 18.5 cm. or over, the conjugate vera must be adequate; on the other hand, an external conjugate under 18.5 cm. does not necessarily indicate an inadequate conjugate vera. However, one must be mindful that in the android pelvis the length of the conjugate vera is no criterion of the capacity of the inlet to allow engagement of the head, because of the angulation of the fore-pelvis.

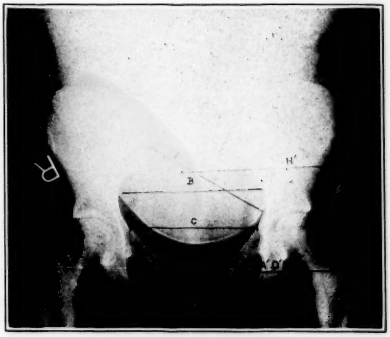

FIGURE 3.

A male type of fore-pelvis preventing the engagement of the head; the anteroposterior and transverse measurements of the inlet were normal.

The work of Jacobs,[9] with use of his inclinometer to measure the anteroposterior diameter of the inlet, has detracted considerably from the value of the diagonal conjugate in determining the diameter of the conjugate vera. On several of our films we have checked the influence of the inclination of the symphysis on the length of the diagonal conjugate, and have found Jacobs's conclusions to be quite accurate.

Anteroposterior Diameter of the Inlet: Conjugate vera

In our cases the anteroposterior diameters of the inlet (conjugate vera) varied from 7.0 to 13.6 cm., the majority being 11.0 cm. or more, as shown in Table 2.

All the cases with a conjugate vera of 9.5 cm. or more (97 per cent) were delivered from below.

TABLE 2. *Length of Conjugate Vera.*

LENGTH	INCIDENCE
	%
11.0 cm. or more	60.5
10.0–11.0 cm.	31.0
9.5–10.0 cm.	5.5
9.0– 9.5 cm.	0.5
8.5– 9.0 cm.	0.5
Under 8.5 cm.	2.0

Of the cases under 9.5 cm., 3 were delivered by cesarean section, 1 by mid-forceps and 2 by normal delivery (Table 3). All cases delivered by cesarean were given a test of labor of at least six

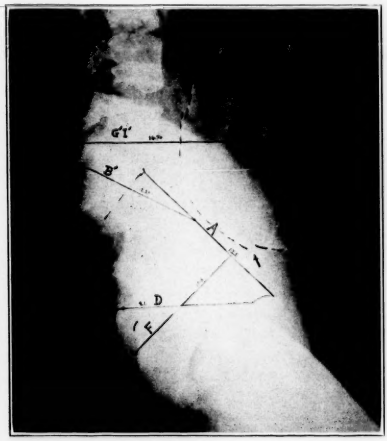

FIGURE 4.

A narrow inlet, with the head molded by uterine pressure in an attempt to engage it.

hours, except a case with a conjugate vera of 7 cm.

The pelves with narrow conjugate veras are usually of the flat or justominor type, and according to Irving,[10] cesarean section is indicated where there is a conjugate vera from 5.5 to 7.5 cm. in flat pelves and from 6.0 to 8.0 cm. in generally contracted pelves. In the flat pelvis, the fact that the head enters the inlet in its transverse diameter may allow the occiput to move toward the side it faces so as to present the bitemporal diameter of 8.0 cm. to the narrow conjugate vera. This occurred in several of our cases. Another way in which the head may enter a narrow anteropos-

TABLE 3. *Data on Cases with Conjugate Vera of Less Than 9.5 Cm.*

LENGTH OF CONJUGATE VERA	TYPE OF PELVIS	METHOD OF DELIVERY	WEIGHT OF BABY
cm.			*lb., oz.*
7.0	Flat	Cesarean	6, 6
8.6	Flat	Cesarean	6, 5
8.4	Flat	Cesarean	7, 11
9.1	Small gynecoid	Mid-forceps	6, 10
8.3	Flat	Normal	6, 7
8.3	Flat	Normal	5, 6

terior diameter is by extreme anterior or posterior asynclitism. Once the head is engaged in the flat pelvis it may have no difficulty in the rest of the

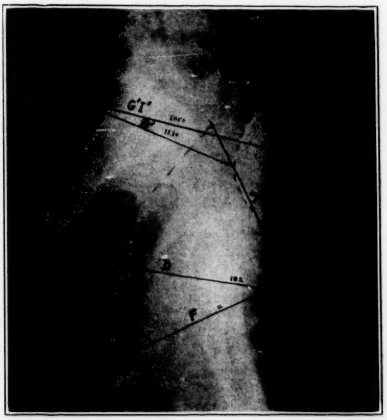

FIGURE 5.

A narrowed, greater sciatic notch, with a short posterior sagittal diameter of the mid-pelvis.

canal, because of a tendency for the canal to increase from the inlet to the outlet. In the justominor or generally contracted type, difficulty in engaging may be followed by further trouble as the head descends, because the small dimensions usually prevail throughout.

The android type offers the greatest difficulty in management. In the latter type the conjugate vera, although apparently adequate according to measurements, may be reduced by as much as 3 cm. in its capacity available for engagement of the fetal head, because of the angulation of the fore-pelvis. Also, due to the usually accompanying funnel type of pelvis, more resistance is encountered as the head descends.

The problem of prognosticating the ability of a certain fetal head to engage in a given pelvic inlet involves more than a consideration of the diameters concerned and cannot be reduced to a mathematical formula. Due consideration must be given to the contour of the inlet, the angulation of the fore-pelvis, the flattening of the posterior pelvis, the encroachment of the promontory on the transverse diameter, the resistance offered by the cervix and its influence in directing the head into the larger or smaller part of the pelvis, the degree of flexion and the moldability of the head, the strength of the uterine contractions and other factors.

In this series we encountered 2 cases, elderly primiparas with adequate pelves, who required cesarean section because of fetal distress after several hours' test of labor, with failure of the cervix to dilate and nonengagement of the fetal head.

Transverse Diameter of Inlet

The transverse diameters of the inlet varied from 10.5 to 14.5 cm., the majority being over 12 cm., as shown in Table 4. The fact that no transverse diameter was under 10.5 cm. suggests that, in our series at any rate, this diameter could not be a cause of dystocia.

TABLE 4. *Length of Transverse Diameters.*

LENGTH	INCIDENCE
	%
13 cm. and over	25
12–13 cm.	49
11–12 cm.	25
Under 11 cm.	1

Where there is a male type of posterior pelvis, with the greatest transverse diameter of the inlet approximating the promontory by less than 3 cm., one must measure the available transverse diameter at least 3 cm. from the promontory; otherwise, one may get a false concept of the roominess of the inlet.

Caldwell, Moloy and D'Esopo[11] have shown that in all types of pelves except the anthropoid 70 per cent of the cases have the head entering the inlet in the occipitotransverse position. Therefore, if a head is well flexed as it presents at the inlet, the suboccipitobregmatic diameter of 9.5 cm. is offered for engagement with a generally adequate transverse diameter of the inlet. This fact renders the biparietal diameter of 9.0 to 9.5 cm. the important fetal diameter to be considered in most cases in relation to the conjugate vera.

Bispinous Diameter or Transverse Diameter of Mid-Pelvis

The bispinous diameter between the tips of the ischial spines, which constitutes the transverse and also the narrowest diameter of the mid-pelvis, varied in our series from 7.6 to 12.5 cm., the majority being 10 cm. or over, as shown in Table 5.

Assuming a diameter of 9.5 cm. to be adequate even for posterior heads, 81 per cent of the cases were in this category. The narrower diameters were found to be causes of dystocia, not per se, but only in association with a narrow posterosagittal diameter of the mid-pelvis and a narrow subpubic arch. The narrow bispinous diameters were found usually in the pelves with an android tendency in the posterior portion of the inlet, and in the funnel-shaped pelves. When the occiput is anterior, the bitemporal diameter of 8.0 cm. presents in this diameter, and consequently in anterior positions the bispinous diameter is invariably adequate. In occiput posterior positions the biparietal diameter presents in the bispinous diameter. If the latter is 9.0 cm. or under, there is a tendency toward an unrotated posterior occiput or mid-transverse arrest, provided there is also a narrow subpubic arch, so that the head, when it reaches the mid-pelvis, may not find room under a wide arch to allow it to recede from the narrow bispinous diameter.

TABLE 5. *Length of Bispinous Diameter.*

LENGTH	INCIDENCE
	%
10.0 cm. or over	56.0
9.5–10.0 cm.	25.0
9.0– 9.5 cm.	10.5
8.5– 9.0 cm.	7.5
Under 8.5 cm.	1.0

Posterior Sagittal Diameter of Mid-Pelvis

The posterior sagittal diameter of the mid-pelvis, which constitutes the width of the greater sciatic notch on a level with the ischial spines, varied from 2.4 to 6.2 cm. Eighty-six per cent of the cases were 3.5 cm. or over, which seems to be normal (Table 6).

The posterior sagittal diameter of the mid-pelvis is important to allow the rotation of posterior occipital presentations. If the occiput is posterior and there is a narrow subpubic arch combined with a bispinous diameter of 9 cm. or less, at least 3 cm. is needed in the posterior sagittal diameter to permit rotation. This type of complication is usually seen in the male types of pelves. For all practical purposes, the posterior sagittal diameter, to be adequate for rotation, should measure at least one third of the bispinous diameter.

In considering the mid-pelvis, one must also be aware of the sacrococcygeal platform and its angulation and relation to the planes of the ischial spines. If the coccyx is markedly angulated at its junction

with the sacrum, or if the concavity of the lower sacrum and coccyx encroaches too much on the plane of the mid-pelvis, a quasi platform may

TABLE 6. *Length of Posterior Sagittal Diameter of Mid-Pelvis.*

LENGTH	INCIDENCE
	%
4 cm. or over	65
3.5–4.0 cm.	21
3.0–3.5 cm.	13
Under 3 cm.	1

be formed to hinder descent of the head, and dilatation of the cervix, with consequent arrest at the mid-pelvis, endangering the fetal cerebrum.

Pubotuberous Diameter

The pubotuberous diameter, suggested by Schuman,[12] which is really the perpendicular length of the fore-pelvis, varied from 6.5 to 10.8 cm. (Table 7). The large majority of cases (73 per

TABLE 7. *Length of Pubotuberous Diameter.*

LENGTH	INCIDENCE
	%
10.5 cm. or over	1
9.5–10.5 cm.	26
9.0–9.5 cm.	38
Under 9 cm.	35

cent) measured 9.5 cm. or less, leaving 27 per cent as potentially funnel in type. The minority usually accompanied the pelves with an android or anthropoid tendency. The pubotuberous measurement is particularly important in cases where a narrow subpubic arch, associated with a long pubotuberous diameter, may present an apparently adequate bituberous or transverse diameter of the outlet. In such cases, measurement of the bituberous diameter alone provides an incorrect conception of the adequacy of the pelvis, owing to the masking of the narrow subpubic arch by the abnormally long pubotuberous diameter. In many cases a wide subpubic arch, with a short pubotuberous diameter, compensates for a narrow bispinous diameter and affords an easy egress for the head.

CONCLUSIONS

The use of x-ray films in pelvimetry has added a more scientific aspect to obstetrics and enables one to prognosticate more confidently the role that the pelvis will play in each obstetric problem. We believe that in the vast majority of cases a test of labor should be given, but the time of the test will vary markedly with the character of the maternal pelvis involved. To perform a cesarean section on the basis of the data obtained from measurements alone, unless the conjugate vera is 7 cm. or less, is to minimize the astounding faculty of Nature to effect delivery from below, even in the presence of apparently insurmountable obstacles. To anticipate an easy labor because of ample measurements is to discount the influence of the cervix, the uterine contractions and the general constitution of the patient in the conduct of labor. Neither the duration of labor nor the probability of operative delivery can be anticipated by consideration of the pelvis alone, because of the variability of the other factors involved. X-ray measurements are but a part of the general picture and to the experienced accoucheur are of distinct value in following the course of labor and helping to indicate when and how to use instrumental intervention.

X-ray pelvimetry should not supplant external and internal measurements and palpation, but should supplement them in questionable cases. Every primipara does not require x-ray pelvimetry, but in the following cases we believe that the procedure is indicated: primiparas with floating heads at term; multiparas with a history of previous difficult deliveries; primiparous breeches, with apparently small pelves by external measurements; cases with narrow subpubic arches and outlets, in order to determine the adequacy of the mid-pelvis; elderly primiparas with external conjugates of 18.5 cm. or less, so that one can more readily differentiate bony from soft-tissue dystocia when the patient is in labor.

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PRACTICAL PSYCHIATRY WITH ADOLESCENTS

II. Technic of Psychiatric Investigation for the General Practitioner

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A RECENT article[1] called the attention of the general practitioner to the problems of adolescence; it emphasized the need for mental hygiene methods in handling these cases. The general survey of the field was outlined, including illustrative case histories, and general recommendations were made as to the part the general practitioner could and should play in helping this type of patient. A surprising number of requests were received for further information and reference material, and several of these inquiries described difficulties that physicians have had with their own children. In view of the apparent need for further elaboration, it has been decided to describe in this paper a technic of psychiatric investigation for the needs of the general practitioner, and in a subsequent paper to describe the technic of psychotherapy, with appropriate case histories, and to suggest a program of reading on the subject of the adolescent and his problems.

GENERAL CONSIDERATIONS

Psychiatric investigation in the adolescent may be entered into at once if the patient comes, or, as more often happens, is brought by the parents, to the physician because of some disturbance of mood or conduct in the home. In general, one should obtain the history first, do a routine physical and neurological examination, and lastly complete the additional history and inquiry concerning the specific problems presented. It is important that the parents be informed of only that amount of interview material that the patient gives the physician permission to discuss with them: one must prevent the patient from developing the idea that his secret confidences have been revealed. Sometimes the case may be discussed privately with the parents if they are exceptionally reliable.

The taking of a psychiatric type of history is, of course, the preferred way to obtain material; it is also essential to cover the field of personality so that no important traits are overlooked. The particular subjects to be covered are stressed in the following section.

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PSYCHIATRIC HISTORY

Persistent *neurotic traits* in children are often early indications of personality deviations. One should therefore question the patient and later the parents concerning bedwetting prolonged after the age of two and temper tantrums, such as episodes of screaming, foot-stamping, breath-holding, assaults on an offending person and destructiveness. The occurrence of these episodes suggests that as a child the adolescent acquired abnormal methods of gaining attention or expressions of sympathy and affection from the parents.

The *day-dreaming and fantasy life,* as well as persistent or vivid night-dreaming, are significant, since they reveal the presence of chronic fear as well as the tendency to withdraw from the practical and real into the attitude of pleasurable and effortless castle-building. This tendency is often the first expression of an introverted or a schizoid personality, and its seriousness is entirely measurable by the amount of time which the adolescent spends in day-dreaming. If along with this activity he shuns physical contacts such as those made in sports, avoids social contacts such as companionships of his age group and prefers to stay by himself and read most of the time, one can conclude that these symptoms of withdrawal are ominous.

Personal ambition, whether practical or impractical, again gives a clue to the personality type as well as to any potential problems of personality. All adolescents go through periods of rather wild ambitions to be famous in a variety of fields of life, and the example of a few famous men in certain fields who from an early age had been obsessed with a desire to follow that particular field makes a proper judgment of an adolescent's choice of goal difficult. For example, given a case in which the adolescent has a strong physique, an extrovert type of personality and a school record showing proficiency in sciences, one would be rather doubtful of his obtaining success in music, particularly if one discovered later in the examination that the father's brother had been a rather well-known musician and that the father had been unconsciously forcing the adolescent in a similar direction. Ambition ideals must be tempered with the practical aspects of the patient's personality

type and background; otherwise, the next few years for that particular adolescent are sure to be excessively difficult.

An accurate description of the adolescent's *loves and attachments,* if it can possibly be obtained, either from him or from outside sources, is of the utmost significance. The absence of proper balance between his attachments to each parent, and the disproportionate interest or attention granted to him by parents, may indicate the basis of feelings of insecurity and emotional tension which one oftentimes sees at this age. In the early years of adolescence one expects the subject's attachment to his own age group to be rather of the unconscious homosexual type, but during the later years, that is, about the age of sixteen and after, his emotional interest should swing to the opposite sex. Tendencies of aggressive or submissive response to overtures of affection need to be properly judged, for here again disproportion in quality or quantity indicates a deviation from the norm. Sorrows and disappointments, depending on their cause and the way in which the adolescent absorbs them or is crushed by them, often indicate the degree of his emotional maturity. If he is excessively depressed by the cold response of an object of his love or by the thwarting of a particular ambition, one should begin planning therapy to promote emotional toughness and maturity. The reaction to the death of a loved one is occasionally quite abnormal in degree, and in this case one should immediately suspect that the patient has been preoccupied with ideas of death and that he may be a definite suicide risk.

Secret fears and uncertainties can cause such emotional disturbance to the adolescent that his efficiency in schoolwork and proper adjustment at home are seriously impaired. One is often surprised at the variety and often the absurdity, from an adult standpoint, of the thing that the adolescent harbors as a secret fear. Strange ideas concerning his family or personal history spring from some half-heard or misinterpreted family gossip, or from a comparison of his own person with an abnormal individual with whom he has come in contact. Some of these ideas may be based on past failures in school, in sports or in social adjustment; magnifying his failures, the adolescent begins to fear that he is abnormal.

Psychic trauma may be either the origin of morbid fears and chronic emotional tension or the precipitating factor of an acute emotional upset. An acutely embarrassing episode in school in which the adolescent is revealed in an unfavorable light to his fellow students, or the occurrence of an expression of fear or cowardice in sports, or the persistence of excessive criticism by his colleagues of some personal physical defect, may form a psychic trauma of sufficient degree to warp the adolescent's adequate adjustment to himself and to others. The commonest source of psychic trauma is in the sexual field, but the unfulfilled desire to be a hero or an object of appreciation and envy by his fellows is also a frequent cause.

Sexual knowledge or misbeliefs, experiences and fears form a considerable portion of the adolescent's personal problems. In present times the amount of sex education available and the method of obtaining it during adolescence are considerably better than they were in the past, yet there is a great deal to be done before this very important life situation can be correctly and safely handled. Despite the apparently early age at which modern boys and girls become acquainted with sexual matters, it is often amazing to discover the amount of ignorance or more often half-ignorance which an adolescent possesses. Because of the excessive attention paid to Freudian concepts from a "popular standpoint," one sees more evidence of a swing of the pendulum in the other direction. One meets discouraging resistance from parents and educators in matters of sex education, and this further suggests that the family physician is much better fitted and in a much better emotional position than the parents or educators to give this information to adolescents. Careful questioning of adolescents often reveals that their seemingly flippant and voluminous acquaintance with sexual topics is nothing but a veneer under which there is considerable uncertainty and a definite groping for accurate information. One has only to have as patients a few adolescents who have been the objects of illegitimate pregnancies, rape, homosexual assaults or venereal disease to realize what tragedies these occurrences can seem to them. On investigating these cases one clearly sees how inadequate is the present amount of sex education that most adolescents receive. It is obvious from occurrences like these that the vast proportion of sexual information is obtained from half-truths described or demonstrated by other adolescents and by vulgar-minded adults. Masturbation has been investigated and described for enough years to be properly understood; yet the majority of lay people and a number of physicians still believe that it is a serious physical and moral offense that casts a shadow on the body, mind and soul of the indulger.

Intrafamily relationships properly evaluated are often indicative of an adolescent's degree of maturity. The defects and immaturity of the only child are well known and often exaggerated, but

they are not exaggerated if the particular adolescent is an only child who is overprotected against contacts with contemporaries. If the only child is permitted to mix with his age group and take the average knocks from his environment, he need not differ in degree of maturity from the adolescent who is one of several siblings. Family relationships that are modified or marred by an excessively dominant parent, by an only child or by a favorite child, may produce in the adolescent's personality definite traits of inadequacy and feelings of inferiority; similar effects are seen when the affection and economic generosity of the parents have been unequally distributed among the children.

The presence of any *symptoms of a psychosis* should be confirmed. Remembering that the early years of adolescence are accompanied by spells of moodiness, vague longings, incompletely formulated desires and ambitions, and short periods of mild restlessness, one can then look for exaggeration of these phenomena and for other abnormalities. Does the adolescent describe difficulty in concentrating or in thinking? Does he feel a sense of strangeness or remoteness within himself? Do his surroundings and the people about him appear unreal? Does he feel that others, including strangers, make fun of him by word or gesture or talk among themselves concerning him? Has he noticed any peculiarity of traits or gestures in those around him to suggest that these have special meaning to him? When alone or at a distance from others has he heard them talking about him, calling his name, making derogatory remarks about him or labeling him with obscene terms? Has he been in strangely close relations with God, either by some sign in his environment or by hearing God's voice? Has his physical appearance changed strikingly in size, contour, color or sex, or has either half of his body assumed a dominant value? In addition, the physician should note in the patient's general appearance and behavior whether he is unduly restless, fearful, suspicious, irritable, silly, manneristic, emotionally flattened and withdrawn, or unresponsive. He should obtain from the relatives a description of the patient's usual conduct or change in conduct, particularly whether he keeps to himself excessively, is careless or excessively careful or fantastic in his personal appearance and personal hygiene, talks to himself, makes odd gestures, shows emotional outbursts or indulges in silly smiling or laughing without adequate cause, seems perplexed, confused or retarded, or, in general, acts or appears strange compared with his colleagues.

Estimation of the Case

First must be considered the acute problem which brought the adolescent to the physician. It may be an unexpected failure in school, or a sudden rebellion against authority, whether parental, religious or civic. Oftentimes it is caused by sexual transgressions, either masturbation or the occurrence of homosexual or heterosexual activity with a playmate. The problem may be the continuance or reappearance of some childhood neurotic trait such as bedwetting or temper tantrums that the long-suffering parents have finally but rather tardily decided needs scientific care. A clash with a sibling or a schoolmate may have resulted in a rather vicious physical reaction, such as assaultiveness or destructiveness. Dishonesty, either lying or stealing, frequently causes the parents to bring the adolescent to the physician for help.

The acute problem is often relatively superficial and only a mask hiding a much more serious, more deep-seated and more prolonged maladjustment that might be termed a chronic problem. In these cases one finds sibling rivalry, resentment or overprotection by parents, depression and uneasiness from guilty feelings concerning masturbation, or unacceptable feelings of physical inferiority in competition with playmates. Perhaps a dislocation of family setting is preying on the adolescent as he finds himself emotionally torn between allegiance to a natural parent and love for a foster or a divorced parent.

Evaluation of the personality type of the patient is possible at this stage of psychiatric examination. By putting together the material obtained from the parents and from the patient and that gained by one's own observation one can accurately classify the personality type as introverted, extroverted, emotionally mature or immature. Subtypes could be included but their inclusion would excessively complicate the procedure from a descriptive, and particularly from a general therapeutic, standpoint. At this stage, too, one must approximate the intellectual capacity of the adolescent, since it is obvious that with a subnormal intellect his maladjustment may be entirely on the basis of bewilderment secondary to demands made on him by family pride and by environment. The labeling of an adolescent as a true psychopath is a serious matter and cannot safely be done by anyone not skilled in psychiatry. The history, physical examination, neurological examination and psychiatric examination may elicit sufficient material to make one suspect a psychosis or a psychopathic state such as postencephalitic behavior disturbance, post-traumatic behavior disturbance, mental deficiency or epileptic phenomena, such as petit-mal attacks, psychic equivalents and automatism.

An appreciable amount of moodiness, poetry writing, abstract philosophizing, chronic cynicism, extreme sympathy for the underdog and so forth

are all quite normal during puberty and adolescence. A tendency to leftist leanings is common in the late period of adolescence and is well demonstrated by the noisy behavior of student groups. The following poems[2] are excellent examples of an adolescent's mental gropings for and understanding of herself, and her environment and the relation of herself to the latter. The authoress was fourteen at the time she wrote the first and seventeen at the time of writing the second.

Death Mood

Is death but a sleeping
From which there is no waking,
A night without a dawn?
Is sleep a taste of dying,
An oblivion of dream-eternity?

What happens in those lethal moments?
Years pass between each living day
Of which we are unmindful . . .
Until slow-creeping Time
Has found us with his cobwebs,
Has bound us with his cobwebs,
Has dragged us to his dungeon
There to shrivel up and die!

Is life but a searching,
A questioning and searching,
A weary futile seeking
For non-existent things?
Is death then the answer,
Is death . . . or God the answer
To the helplessness, the hopefulness,
The beating of our wings?

Death Mood (later)

I

Since you must seek me out, oh, peaceful death,
I pray that you will come on velvet feet
Into my weary heart, and, for a breath,
Feel its glad welcome in a quickened beat.
Or, as I lie upon these sunlit sands,
Beckon to me across the ocean wave
That I may swim to your outstretching hands,
Rather than join you in some earthy grave.
But spare me an oblivion too deep
Until I write a final, brief farewell
To my few friends. Then, as I fall asleep,
Let the bewitching solo of a bell
That I have often held my breath to hear,
Sound its enchanting music in my ear.

II

Since I must die, I ask that death may be
A quick ecstatic moment when it comes.
I do not wish to feel security
Slip from me slowly, nor the peace that numbs
Quiet my throbbing pulse. No chair for me
On a sunny lawn and a nap at noon!
I have pursued a life so bold and free . . .
Rather than creeping age, let death come soon! —
A dagger thrust that swiftly finds it mark,
A sudden leap into a rushing stream,
Or stumbling footsteps on a cliff at dark,
Would capture death outside some tortured dream;
Nor let me falter at the black void's brim,
Fearing what may be seen beyond the rim!

(Four additional stanzas follow.)

Conclusion

A practical mode of psychiatric investigation applicable to handling the problems of adolescence is outlined, and a brief explanation of the various etiologic factors met with in such cases is made. This age group of patients, with the clinical picture peculiar to them, forms a segment of potential patients who merit the serious attention of the general practitioner. By the application of the principles of mental hygiene not only can the family physician help some of these boys and girls over difficult spots during their adolescent years, but he can very frequently abort any tendency to the development of a neurosis, psychosis or warped personality. When he is giving periodic care to the children, the alert family physician can suggest to the parents that at puberty these young people be referred to him for definite sex education, and that during the later years of adolescence they again seek his counsel on other problems of personality adjustment.

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AN EXPERIMENTAL STUDY OF THE TREATMENT OF AIR EMBOLISM*

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THE increasing usefulness of air as a contrast medium in radiological diagnosis requires adequate preparation to deal with the one major catastrophe which may result, namely, air embolism. Circulatory occlusion by air may be disastrous in three ways: by obstructing a sufficiently large area of the pulmonary capillary bed to interfere with the transfer of an adequate volume of oxygenated blood from the right ventricle to the left auricle; by obstructing one or more branches of the coronary arteries; or by rendering ischemic a vital area in the brain. If death does not result at once, the outcome will depend on the function of the involved area and the length of time it can tolerate ischemia. Since time is of the greatest importance, immediate steps to absorb the nitrogen must be undertaken to mitigate the serious effects which not infrequently cause death later. In several publications[1–5] from this clinic, experimental and clinical evidence was provided to show that the absorption of nitrogen from the body tissues is facilitated by the inhalation of 95 to 100 per cent oxygen. The application of this agent was recommended among other things for the treatment of air embolism,[5] although no clinical opportunity to test such a situation had presented itself. Meanwhile, clinical accounts of air embolism continue to be published,[6, 7] which demonstrate the helplessness of the physician in such accidents, although the present practice of decompressing divers with oxygen, as suggested by Behnke et al.,[8] should provide a broad hint as to what to do.

To re-emphasize the value of immediately applying pure-oxygen inhalations for air embolism, we have carried out a series of simple experiments on rabbits, instead of waiting for a personal clinical experience to demonstrate its effectiveness.

METHOD

The right or left carotid artery of 9 rabbits was exposed under ether anesthesia. Three to 5 cc. of blood was withdrawn, mixed by shaking with 1 cc. of air and reintroduced into the artery. The artery was then ligated to prevent bleeding, and the wound was closed. In nearly every experiment a tetanic spasm of the entire musculature of the body resulted immediately, but invariably recovery occurred promptly. In Experiment 3 (Table 1), immediate death resulted because the air was insufficiently dispersed into tiny bubbles during the mixing with blood. On recovery from the anesthesia, paralysis of one or more muscles in one or more extremities was observed, and the length of time for return of normal function was noted.

A second series of 13 rabbits was treated and observed in similar fashion, except that immediately following the air injection the rabbit was placed in an oxygen chamber (90 to 100 per cent concentration), in most cases for one or two hours. The first two rabbits, exposed for three and eight hours respectively, succumbed to pulmonary edema caused by the intolerance of rabbits to high concentrations of oxygen for more than one-hour or two-hour periods.

RESULTS

Damage to muscular function of the extremities occurred in all but 2 of the 21 animals surviving the air injection. The determination of the extent of muscular paresis was possible only after recovery from ether anesthesia, except in Experiments 20, 21 and 22, in which local anesthesia was used. Since there were no simple objective signs of air embolism except muscular paresis, we utilized this as a measure of the extent and duration of injury caused by the air embolus for the purpose of evaluating the therapeutic effect of breathing pure oxygen.

Table 1 demonstrates clearly the considerable advantage that oxygen provides. The 8 surviving rabbits in the control series required an average of *eleven days* for full recovery from muscular paresis. In contrast, 10 of the 11 rabbits that showed muscular weakness and were given oxygen inhalations required an average of less than *ten hours* for full recovery of function. In the eleventh rabbit (Experiment 22) oxygen was apparently of no benefit, and death resulted on the fifth day. The data in the "Comment" column of the table include details that emphasize the sharp difference in the course of events induced by the oxygen inhalations.

The two rabbits that showed no apparent injury following the air injection (Experiments 16 and

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TABLE 1. *Data on Rabbits.*

Experiment No.	Carotid Artery Injected	Immediate Effect of Air Embolism	Time Required for Full Recovery	Comment
CONTROL SERIES				
1	Right	Marked paralysis of all extremities	10 days	1 cc. blood mixed with 1 cc. air; hindlegs normal after 48 hours; left foreleg normal on 5th day; extensor paralysis of right foreleg last to disappear.
2	Right	Extensor paralysis of right foreleg	13 days	2 cc. blood mixed with 0.75 cc. air; no substantial change for 12 days.
3	Right	Immediate death	—	3 cc. blood and 1 cc. of air, poorly mixed.
4	Right	Severe paralysis of all extremities	14 days	Slight improvement in all legs on 3rd day, but could not jump or sit up; hindlegs normal and right foreleg slightly improved on 8th day; extensor of left foreleg still slightly paretic on 12th day.
5	Right	Extensor paralysis of right foreleg	5 days	Improvement not evident until 4th day.
6	Right	Extensor paralysis of both forelegs	12 days	Left foreleg normal after 24 hours; some improvement of right foreleg on 8th day.
7	Right	Complete paralysis of all four legs	13 days	Both left limbs improved on 3rd day and almost normal on 6th; on 8th day only right foreleg extensor paretic.
8	Left	Complete paralysis of right hindleg, moderate paralysis of both forelegs	12 days	Slight improvement of both forelegs after 24 hours; right foreleg normal on 4th day; both forelegs normal on 7th day.
9	Left	Paralysis of all four legs, most marked in left foreleg	7 days	Hindlegs improved within 24 hours and normal on 4th day; extensor of left foreleg still markedly paretic until 6th day.
OXYGEN SERIES				
10	Right	Extensor paralysis of right foreleg	4–16 hr.	Breathed pure oxygen for 8 hours; death on 3rd day with edema of right lung and right hemothorax; extensor paralysis showed improvement after 4 hours in oxygen tent and was entirely absent when observed the following morning.
11	Right	Extensor paralysis of right foreleg	5½ hr.	Breathed pure oxygen for 3½ hours; removed from oxygen because of increasing dyspnea; no paralysis 2 hours later, but progressive dyspnea and death in 22 hours; autopsy showed bilateral pneumonia, "wet lung" and retrobulbar hemorrhage.
12	Right	Marked paralysis of all four legs, completely immobilized	3½ hr.	Removed from oxygen tent after 2 hours; ½ hour later showed marked improvement; jumped about without disability 3½ hours after air injection.
13	Right	Severe extensor paralysis of left foreleg and slight paralysis of left hindleg	2–18 hr.	Marked improvement of left foreleg and complete recovery of left hindleg within 2 hours after air injection; fully recovered following morning.
14	Right	Slight paralysis of both forelegs	1–17 hr.	Left foreleg normal, jumped and sat up well within 1 hour after air injection.
15	Left	Slight weakness of right foreleg	1½ hr.	
16	Right	No effect	—	
17	Right	Marked paralysis of right foreleg	3–20 hr.	Recovery almost complete 3 hours after air injection; complete next morning.
18	Left	Slight paralysis of right foreleg	2 hr.	Recovery almost complete after 2 hours; death in 17 hours; autopsy showed hemothorax.
19	Right	No effect	—	Showed no tetanic spasm after injection.
20	Right	Marked paralysis of both forelegs	2–18 hr.	Experiment under local instead of ether anesthesia; given 100 per cent oxygen by tracheal cannula for ½ hour preceding air injection; typical convulsive seizure and paralysis; left foreleg normal after breathing pure oxygen for 2 hours; right foreleg still a bit weak, but normal following morning.
21	Right	Complete paralysis of all extremities and of posterior neck muscles	—	Experiment under local instead of ether anesthesia; given 100 per cent oxygen by cannula for ½ hour before and 1 hour after air embolism; 5 hours later hindlegs slightly improved; much better after 48 hours, but paralysis complete in right foreleg and severe in left foreleg on 4th day; died on 5th day of pneumonia and basilar meningitis.
22	Right	Severe paralysis of all four legs	24–42 hr.	Experiment under local anesthesia; given 100 per cent oxygen by tracheal cannula for ½ hour before and 1 hour after air injection; within 2 hours left foreleg and hindlegs normal; after 5 hours only extensor paralysis of right foreleg; after 24 hours very slight weakness of right foreleg, which was normal the following morning.

19) were among those given oxygen immediately after the air injection. One of these showed the characteristic convulsive spasm observed immediately following the injection of air into the carotid artery. In this case the failure of muscle weakness to appear might have been due to an unusually rapid absorption of the nitrogen by the oxygen inhalations. In the other case, however, a convulsive seizure did not follow the air injection, so that the absence of muscle weakness on recovery from the anesthetic cannot be considered a result of the administration of the oxygen.

In Experiments 20, 21 and 22, 100 per cent oxygen was given by tracheal cannula for half an hour prior to producing the air embolus in order to test the prophylactic value of this agent. All animals developed muscular paresis. The prophylactic value of oxygen was not demonstrated, since the time for full recovery in the 2 surviving rabbits (Experiments 20 and 22) was not less than that for the rest of the group, in spite of the additional inhalation of oxygen following the air injection. This observation is in agreement with that from a similar study previously made in cats, in which no protection against air injected intravenously was afforded by a preliminary period of breathing pure oxygen.[1]

Discussion

The time required for a bubble of nitrogen blocking a cerebral vessel to diffuse out of the blood stream into the expired air while 100 per cent oxygen is being breathed depends on the size of the bubble, but it is probably completely disposed of within the time of exposure to oxygen utilized in these experiments.[1] It was not possible to determine whether the time for recovery of function might have been shortened still further by a longer exposure to oxygen, because of the intolerance of rabbits to longer exposures. The relatively long duration of the paresis as compared with the short time of exposure to oxygen is clearly due to the long period of recovery required by the injured nerve cells. The more rapid recovery of function by the nerve cells of the rabbits that were given oxygen is not adequately explained on the basis of a greater oxygen saturation of the brain, for the oxygen deficit produced by the air embolus is probably far greater than can be compensated for by an increase in diffusion of oxygen from neighboring tissues. The mechanism of the action of oxygen in these circumstances is therefore, in our view, that referred to in a preceding publication[9]: that is, the inhalation of pure oxygen necessarily and rapidly reduces the pressure of nitrogen in the lungs toward zero, so that nitrogen in the blood, which is then at a relatively much higher pressure, must, according to Henry's law, diffuse into the expired air.

The technic of the procedure in these experiments included ligation of the carotid artery following air injection. This was found necessary because the needle-puncture wound tended to bleed in spite of pressure for several minutes. Ligation of one carotid artery in the rabbit does not cause muscular paralysis. It therefore cannot be of consequence in evaluating the benefit of the oxygen, since the same technic was applied to both series of animals.

The failure to observe any benefit from the inhalation of 100 per cent oxygen in advance of the production of the air embolus is presumably due to the fact that the flow of nitrogen from the blood stream to the alveolar air is rapidly slowed because of the immediate rise in the partial pressure of nitrogen in the lungs as soon as the first breath of air replaces that of pure oxygen.

The use of 100 per cent oxygen for the treatment of human cases of air embolus cannot be expected to show the remarkably uniform success obtained in these experiments, even if applied without delay at the instant air embolism occurs. This should be obvious from the fact that much depends on the volume of the air embolus. The amount of air which can be tolerated without serious consequences by the pulmonary capillary bed is obviously much greater than it is in the brain or the coronary arteries. The entrance of air into the latter is not likely except in the case of a patent foramen ovale or when, during a thoracic procedure, air enters a tributary of a pulmonary vein. Although air may reach the brain under similar circumstances, an alternative route of cerebral air embolism may be that recently demonstrated by Batson,[10] that is, the vertebral venous plexus, which by virtue of its rich anastomosis with the tributaries of the vena cava and azygos veins may act as a by-pass of the systemic venous circulation. By this route, however, obstruction of the cerebral circulation by air would require the existence of arteriovenous anastomoses, the evidence for which is equivocal.[11] On theoretical grounds a high degree of success should attend the use of 100 per cent oxygen inhalation when small emboli reach the coronary arteries or the brain. Even a large embolus, if not immediately fatal, might be absorbed in time to avert permanent damage or subsequent death.

The substitution of oxygen for air as a visualizing agent does not prevent the adverse effects of gases in body tissues or cavities.[1] This observation

has recently been confirmed by Moore and Braselton.[12]

The very rapid ascent into high altitudes by military aviators using interceptor planes is said to produce symptoms due to the liberation of free nitrogen in the blood stream. The evidence from these experiments and previous publications[1, 2, 5, 8] indicates that such symptoms can be minimized or avoided altogether by pre-oxygenation with 100 per cent oxygen and by the continued inhalation of 100 per cent oxygen during ascent. Pre-oxygenation would remove the nitrogen dissolved in the blood stream, and the continued inhalation of oxygen during ascent would cause the nitrogen entering the blood stream from the tissues to escape via the lungs without forming gaseous emboli.

Conclusions

In rabbits the serious effects of an air embolism that was not immediately fatal were prevented or rapidly alleviated by the prompt administration for several hours of 100 per cent oxygen inhalations. This procedure may be of significance in the treatment of certain types of human cases.

In rabbits the use of 100 per cent oxygen inhalations as a prophylactic agent against air embolism had no beneficial effect. The same would probably hold true in patients when air is injected for diagnostic purposes.

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GLOMUS TUMOR

Report of a Case

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TUMORS of the glomus[1, 2] or arteriovenous anastomosis of the cutis are rare, but are striking because of their symptomatology — chiefly intense pain on pressure and a satisfactory response on local excision. The tumors are usually single, rarely multiple[3] and always benign. One of the frequent locations is beneath the nails. A fairly typical case is reported below.

Case Report

An unmarried, white, 28-year-old French-Canadian woman was seen on November 5, 1938, complaining of having been tired and sleepy for the last 2 years and of having lost interest in people and in life in general. She stated that 2 years previously she had had a nervous breakdown, which she attributed to overwork.

When questioned as to how she rested at night, she admitted that she was awakened several times by severe pain in the middle finger of the left hand, initiated by the slightest pressure or motion during sleep. During the day, she had to perform her work with four fingers of the left hand while she kept the middle finger extended to avoid the slightest jarring or pressure. Any rather severe blow or squeeze caused extreme pain, mostly in the terminal phalanx, but also radiating up the left hand and forearm.

The patient was not interested in the finger, but insisted that she had come for a tonic. She believed that nothing could be done for the finger, since many doctors during the previous 15 years had attempted to make a diagnosis and treat the condition, without success. X-ray examination had been negative. Her case had been thoroughly studied in large cities of New England, and at one time she had obtained partial relief from pain by alcohol injection at the base of the affected finger.

She was told that the painful finger was the cause of her fatigue, nervousness and pessimistic attitude toward life. Physical examination was entirely negative except for the exquisitely tender finger. By pressure with a blunt probe tenderness could be shown to be limited to an area about 5 mm. in diameter on the palmar side of the terminal phalanx. There was no visible swelling or redness, and nothing could be felt superficially on palpation. Pain and tenderness would not permit firm palpation, which might have revealed a small mass below the fat pad.

The symptoms were so suggestive of glomus tumor that, even in the absence of demonstrable tumor, exploration of the finger was advised. This was done under procain anesthesia on November 19, 1938, at Jordan Hospital, Plymouth, Massachusetts. Below the fat pad, lying almost on the anterior surface of the bone, a firm, spherical tu-

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mor mass 4 mm. in diameter was carefully dissected so that a fine blood vessel of entry at the proximal pole and a similar vessel at the distal pole were demonstrated. Very fine nerve fibrils were seen to spread out toward the tumor. The blood vessels of entry and exit were tied off with fine catgut, and the tumor excised.

The next day, the patient stated that the soreness from the incision was not nearly so bad as the previous pain and tenderness that she had endured almost every day for the preceding 15 years.

The pathological description by Dr. Shields Warren was as follows:

> A roughly spherical, brown, moderately firm piece of tissue about 0.4 cm. in diameter. Section shows a discrete tumor partly surrounded by a fibro-fatty capsule. There are several myelinated nerves and arteries in the capsule. There are also nerves forming part of the walls of vascular sinuses. The tumor proper is composed of quite discrete clumps of epithelial-like cells. These cells are compactly arranged and characterized by medium-sized finely granular, round to oval nuclei and indefinitely outlined cytoplasm that appears to merge with that of neighboring cells, forming a syncytium. In some of these cell masses there are elongated nuclei lying in a bed of finely fibrillary tissue. These structures resemble nonmyelinated nerve fibers and merge imperceptibly with the matrix in which the cell groups lie. This matrix is in part a fine fibrillary substance and in part a homogenous-staining hyalin-like substance, in which may be seen sparsely scattered slender oval nuclei, vascular channels lined by endothelium and often without a definite wall, nonmyelinated nerves and foci of myxomatous degeneration. The most interesting feature of this matrix substance is the close association of nerves and vascular channels, which, as noted in the capsule, are often united so that the nerve appears to form an essential part of the wall of the artery. Diagnosis: glomus tumor.

FIGURE 1. *Section of Glomus Tumor.*

The patient has had no recurrence of pain or tenderness. Her outlook on life has become quite cheerful, and she is energetic and in the best of health. She was married in July, 1940.

Even though glomus tumor is rather rare, the importance of its recognition by the medical profession is obvious. Some patients, after years of torture, have contemplated suicide. Their suffering can have a profound effect on their lives. They have been given diagnoses of neuritis, neuralgia or neurasthenia.

Glomus tumor is a vascular tumor composed of a varying amount of epithelial or fibrous tissue and very sensitive nerve endings or of a characteristic blending of nerve tissue in vascular tissue. It is usually quite small, 2 to 10 mm. in diameter, and is found subungually in the extremities or subcutaneously over various parts of the body. Under the nail or under the skin it appears as a reddish-blue spot. Excruciating pain caused by the lightest pressure is the characteristic symptom. Glomus tumor has been descriptively called neuroangiofibroma.

SUMMARY

A case of glomus tumor is reported which was unusual in its location and in that, except for extreme tenderness, no physical signs of its presence could be ascertained before operation.

1 Carver Street

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REPORT ON MEDICAL PROGRESS

CLINICAL AND EXPERIMENTAL EVIDENCE ON THE NUTRITIONAL REQUIREMENTS IN OBSTRUCTIVE JAUNDICE*

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OWING to advances in preoperative and postoperative care, it has become possible to perform remedial or palliative surgical operations on desperately sick jaundiced patients without undue risk. Recent biochemical studies have thrown light on some of the complicated metabolic errors that occur in obstructive jaundice and other forms of liver disease. As the nature of the hepatic impairment comes into clearer focus, preoperative needs grow less obscure, and the cause of grave postoperative complications may be corrected. An impressive illustration is the discovery of vitamin K and the solution of the problem of the dangerous bleeding tendency in obstructive jaundice, an achievement dependent on the joint efforts of biochemists and alert clinicians. Recognition of the clinical fruitfulness of knowledge gained in biochemical and physiological studies leads the resourceful clinician to give attention to the teachings of the laboratory.

For over sixty years it has been known that the hepatic glycogen content falls in animals exposed to such poisons as chloroform, arsenic and phosphorus, and that simultaneous increase in hepatic lipid occurs. Such observations led to studies of the effect of diet on hepatic reserve and on hepatic composition. Various investigators have shown that preliminary feeding of carbohydrate reduces the susceptibility of animals to hepatotoxic agents, whereas feeding of fat has the opposite effect.[1–5] Opie and Alford[2] found, for example, that three times the oral dose of chloroform fatal for white rats receiving a fat diet is tolerated by animals on a meat diet, whereas five times this dose may be safely given to animals that have been fed on starch and sugar. The protective effects of high-carbohydrate feeding and the harmful effects of dietary fat have been shown to hold in the presence of hepatic trauma produced in various ways, as by ligation of the common duct, subtotal hepatectomy, formation of an Eck fistula and production of biliary cirrhosis by cholecysterostomy.[6, 7]

The manner in which abundant glycogen stores protect the liver from noxious agents has been the subject of considerable speculation. Rosenfeld,[1] noting the reciprocal relation between stores of glycogen and fat in the liver under a variety of conditions, concluded that fat transported to the liver accumulates there as a secondary phenomenon in the absence of sufficient carbohydrate for its combustion. In his view lack of glycogen is the controlling factor, for without a supply of readily available fuel, the detoxifying and reparative functions of the liver are crippled. There appears to be no evidence, however, that the oxidative processes of hepatic tissue undergo an increase in poisoning. Wells[8] suggested that hepatic glycogen by its antiketonic action tends to maintain cell neutrality, thus preventing acidosis and degeneration of hepatic tissue. Opie and Alford[2, 3] favor the hypothesis that a high-carbohydrate intake protects against fat-soluble hepatotoxins, such as chloroform and carbon tetrachloride, by minimizing the deposition of fat in hepatic and renal parenchyma, with a consequent reduction in the amount of the toxin taken up by the tissue. The following experimental observations indicate the fact that the nutritional state of the liver and resistance to hepatic trauma are less simply related than this hypothesis suggests. A high-carbohydrate diet is protective in hepatic trauma produced by ligation of the common duct and subtotal hepatectomy; a high-protein intake protects against chloroform poisoning even when hepatic fat content is high[9]; the effects of phosphorus poisoning are heightened in rats by a meat diet rather than fat.[2]

The well-known protein-sparing action of carbohydrate has been invoked to explain the protection that abundant hepatic glycogen stores afford against hepatic injury. In studying this question Davis, Hall and Whipple[10] obtained convincing evidence of such action in chloroform poisoning after the animals had been fed carbohydrate heavily. They concluded that there is a true conservation of split products of hepatic protein, and that an abundance of glycogen leads to resynthesis of partially disintegrated protein from its original elements. In other words, protein-sparing occurs not at its source, but as conservation of protein end products. The work of Smith and Moise[11] on

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the regeneration of liver tissue after chloroform poisoning yielded data consistent with this interpretation. Further study is needed on the relation between the chemical composition of the liver and its vulnerability if this important question is to be elucidated. Such investigation must take note of differences in response of different species, the role of accessory food factors as well as the cardinal foodstuffs, variations in defense reactions to different kinds of poisons and to mechanical trauma or infection, and the possible significance of renal function in influencing hepatic resistance. The distinction between degeneration and repair should be kept in mind, for the ability of hepatic tissue to withstand a single trauma may not parallel its power to heal.

In view of the necessity of conserving the hepatic glycogen stores, studies of the effects of hepatic disease on glycogenesis and glycogenolysis are of great interest. The work of Mann[12] and Soskin[13] has made it clear that the liver is the sole source of the glucose in the blood. Under physiologic conditions the concentration of glucose in the blood is stabilized by the liver, and an increased intake of glucose leads to glycogen formation and storage, whereas depression of the blood-glucose content is combated by glycogenolysis and liberation of glucose from the liver. The sensitivity of this vital homeostatic mechanism is impaired by hepatotoxins in the experimental animal,[14–16] and by chronic hepatic disease in man.[17–20] Disturbances in glycogenesis, glycogenolysis and gluconeogenesis may result. Although higher concentrations of glucose in the blood may be necessary to effect hepatic glycogen storage, the damaged liver is usually still capable of glycogenesis.[21] The evidence indicates that disturbances in hepatic function leading to hepatic insufficiency may be accompanied by high, low or abnormally labile blood-sugar values, but that glycogen storage in the disabled liver still occurs if sufficient quantities of carbohydrate are taken.

Whether glycogen deposition in the disabled liver proceeds more rapidly when glucose is given by vein than when sufficient carbohydrate is taken by mouth is an unsettled question.[22] The intravenous administration of glucose solution provides a strong immediate stimulus to glycogenesis, and often makes it possible to give larger amounts of glucose than the sick patient can take as carbohydrate by mouth. It is true that hypertonic glucose solution must generally be used. The consensus opposes the use of insulin during intravenous glucose therapy in the nondiabetic patient, and as Soskin and Hyman[23] point out, insulin may even be harmful. The injection of insulin, according to them, causes increased storage of glycogen in the muscles, but without corresponding increase in the liver. Such peripheral glycogen formation tends to lower blood-glucose concentration and thus to reduce the stimulus to hepatic glycogenesis.

Demonstration of the beneficial effects of high-carbohydrate intake in the experimental liver disease of animals has led to the clinical application of carbohydrate therapy in liver disease, with gratifying results. Jones[24] was able to show a sharp reduction in the mortality rate in patients with acute hepatic insufficiency after the introduction of intravenous glucose therapy and a high-carbohydrate diet. He concluded that the more intensive the intravenous glucose therapy the better the prognosis. Ravdin and his co-workers[25] found that the postoperative mortality rate in patients with obstructive jaundice was cut in half with the routine administration of glucose intravenously before and after operation. Later Ravdin[26] expressed some skepticism as to the practical value of intravenous glucose therapy and stressed the use of a high-protein, high-carbohydrate diet in preparing the patient with obstructive jaundice for operation. The value of giving glucose solutions freely by vein in the treatment of liver disease is attested by the experience of various other clinics.[21, 27, 28]

Increasing experimental evidence points to a relation between the protein stores of the liver and its ability to recuperate from trauma. In work[9] already referred to it was noted that a high-protein diet produced in rats maximal resistance to chloroform poisoning, regardless of the lipid content of the liver. Miller and Whipple[29] observed in dogs an increase in liver damage from chloroform anesthesia as the protein stores were depleted, as by bleeding, plasmapheresis or a low-protein diet. A single large feeding of protein thirty-six hours before chloroform anesthesia seemed to be followed by distinct diminution in the extent of liver damage. Messinger and Hawkins[30] found a high-protein diet even more effective than a high-carbohydrate diet in preventing liver damage from arsphenamine. Recently, in a study of dogs with ligated common ducts, maximal glycogen and minimal fat deposition was found[31] to result from a diet high in protein as well as carbohydrate. Brues, Drury and Brues[32] have shown that regeneration of hepatic cells after subtotal hepatectomy proceeds fastest on a high-protein diet, their work being confirmatory of the earlier conclusions of Moise and Smith.[33]

In connection with the composition and functional reserve of the liver as influenced by diet, the work of Best and his colleagues[34, 35] and of

Channon and Wilkinson[36] is of great interest. These workers showed that the extreme fatty infiltration of the liver that occurs in depancreatized dogs given insulin could be prevented by feeding lecithin, choline, betaine, casein and other dietary proteins. Dragstedt and his associates[37] demonstrated that fatal fatty degeneration and infiltration in the liver could also be prevented by feeding raw pancreas or an alcoholic extract of beef pancreas. The existence of a pancreatic fat-metabolizing hormone was announced, and the new hormone was named "lipocaic." MacKay,[38] in confirmation of this work, reported that pancreatic extracts made by Dragstedt's method prevented the deposition of fat in the livers of rats fed a diet deficient in choline and other lipotropic elements. Best and Ridout[39] have questioned Dragstedt's conclusions, and consider that the lipotropic effect of pancreatic extract is not that of a new specific factor, but is due to the choline and protein content of the extract. In a later report, Dragstedt[40] seems to have shown that the choline content of the pancreatic extract he produced was entirely too small to explain its lipotropic power.

A large store of protein material is found in the normal liver,[41, 42] and as Whipple points out,[43] all the evidence indicates that the liver is of primary importance in protein metabolism. It has been shown[44] that after a rapid and extreme plasma depletion the dog can throw into the circulating plasma considerable amounts of protein within an hour or two. The presence of an Eck fistula or of severe liver injury abolishes this reaction.[45] A fall in plasma protein concentration with alteration of the albumin-globulin ratio can be produced in dogs by repeated oral doses of carbon tetrachloride.[46]

It is a common clinical observation[47–50] that the plasma protein concentration may be low in various types of hepatic disease, and that the albumin fraction is more often depressed than the globulin fraction. Butt, Snell and Keys[51] showed that determination of total serum protein concentration may fail to disclose sharp disturbances in the serum protein pattern in liver disease, and that the relation between serum protein concentration and colloid osmotic pressure is variable, owing to changes in the albumin-globulin ratio. These authors in studying 15 cases of obstructive jaundice found an average serum protein value of 6.2 gm. per 100 cc., an average albumin-globulin ratio of 1.22 and an average serum colloid osmotic pressure of 250 mm. of water, the average normal value for the latter being 370 mm. These findings illustrate the point that although the total plasma-protein concentration is important, of greater significance are changes in colloid osmotic pressure and alterations of the plasma protein pattern.

The implication that such experimental and clinical observations have on the treatment of patients with severe liver disease is clear concerning the great value of transfusions of blood and plasma, but as concerns the indications for a high-protein diet the conclusions are less clear-cut. It has long been known that dogs with Eck fistulas can be maintained in reasonably good health only if the meat content of the diet is kept low. Opie and Alford[2] found that phosphorus was more toxic in rats kept on a meat diet than in those kept on a fat diet. Mann[7] was able to keep dogs with obstructive jaundice, cirrhosis or a surgically reduced hepatic mass in fairly good condition for many months, but on the substitution of meat for carbohydrate in the diet, ascites and liver failure set in. Aqueous meat extracts, free from protein and fat, were even more harmful to hepatic reserve.[6] Apparently in considering the influence of a high-protein diet on the liver, the distinction must be made between protein of meat and of milk, for the experimental evidence from various sources[6, 9, 33] indicates that casein has beneficial effects. Although there can be no doubt that feeding casein to the healthy animal increases resistance to experimental hepatic trauma, it still remains to be shown that the damaged liver responds favorably to a high casein intake, and for the clinician this is an important distinction. A reasonable course in the treatment of hepatic disease at present seems to be the inclusion of moderate amounts of protein in the diet, with a preference for the protein of eggs and milk.

The liver in its role as "the commissariat of the body"[52] plays a leading part in the metabolism of the vitamins, although much remains to be learned about the mechanisms involved. The experimental and clinical evidence turns on such questions as changes in hepatic function and morphology due to vitamin deficiency, interference with vitamin storage in liver disease, failure in absorption of vitamins or their precursor substances and impaired conversion of precursor material into the metabolically useful factor.

György and Goldblatt[53] found that rats fed a basal diet lacking in the vitamin B complex and supplemented by thiamin chloride, riboflavin and pyridoxin during the course of four to twelve weeks developed fatal degenerative and necrotic changes in the liver. The hepatic lesions were of the acute and precirrhotic type seen after carbon tetrachloride poisoning, and the changes did not occur if yeast or Peters's eluate was included in the diet. However, the ration used was almost devoid of purine substances, which left the nature of the

nutritional defect uncertain. The authors comment on the chemical and toxicological studies of Neale and Winter,[54] who offered some evidence suggesting that sodium xanthine and some other related purine derivatives, including guanosine, guanine and hypoxanthine, exhibit protective action against the hepatic lesions due to chloroform or carbon tetrachloride poisoning.

Rich and Hamilton[55] were able to produce cirrhosis of the liver consistently in rabbits by giving them a basal diet supplemented with various vitamins but lacking yeast. The evidence pointed to the conclusion that the cirrhosis, which was of the human Laennec type, was due to the lack of some factor contained in yeast but different from thiamin chloride, riboflavin, nicotinic acid and pyridoxin. The possibility that lack of choline in the diet may have produced the hepatic changes was not excluded by the experimental data, but as the authors point out, fatty infiltration rather than portal cirrhosis is to be expected from choline deficiency.

In a study of the vitamin A content of hepatic tissue obtained at autopsy in over 1000 cases, Moore[56] found that cirrhosis of the liver was one of the diseases showing the lowest values. He concluded that the hepatic storehouse may undergo vitamin A depletion in various diseases. Stewart and Rourke[57] have noted the frequent occurrence of low or even absent vitamin A concentration in the plasma of patients suffering from obstructive jaundice. The concentration of carotenoids was less frequently and less severely reduced, a finding plausibly explained by failure of conversion of the precursor substances in the disabled liver. Depletion of vitamin A in the liver and in the plasma is to be expected when the amount of cholic acid delivered into the gastrointestinal tract is deficient, as in obstructive jaundice or impairment of the synthetic functions of the liver, for the vegetable carotenoid substances as well as vitamin A are fat-soluble. In studying the rate of absorption of vitamin A from the gastrointestinal tract in 21 patients with catarrhal jaundice, Breese and McCoord[58] demonstrated greatly retarded absorption in 12 patients. The delay in absorption was roughly proportional to the liver damage, as measured by the bromphalein test, but not to the degree of bilirubinemia.

Vitamin D, like vitamins A, K and E, is fat-soluble, and reduced absorption from the gastrointestinal tract is to be expected in chronic liver disease, obstructive jaundice and biliary fistula. Skeletal decalcification, based on inadequate absorption of vitamin D and calcium, has long been known as a consequence of prolonged loss of bile in dogs with biliary fistulas. Greaves and Schmidt[59] showed that vitamin D is not absorbed from the gastrointestinal tract of rats in the absence of bile salts. McNealy, Shapiro and Melnick,[60] recognizing the possibility of vitamin D lack in jaundice and erroneously assuming its relation to the bleeding tendency, reported good results from the administration of viosterol in jaundiced patients. Stewart and Rourke[61] found abnormally low plasma vitamin C values in patients with untreated obstructive jaundice, and noted that normal values could be established before operation by giving from 300 to 600 mg. of vitamin C daily.

Good results in the treatment of alcoholic cirrhosis of the liver by nutritional therapy have been reported,[62, 63] and the value of the vitamin B complex has been stressed. Whether the nutritional defects in these cases are due entirely to an inadequate diet, whether the disease of the liver contributes to the malnutrition and whether the nutritional deficiency favors the development of cirrhosis are questions for further investigation. The study[55] previously referred to should be noted, in which cirrhosis of the liver closely resembling human portal cirrhosis was produced in rabbits by feeding them a diet adequate except for certain components of the vitamin B complex. Annis and Comfort[63] state that increasing clinical experience indicates that the vitamins, especially thiamin and the vitamin B complex, are beneficial in treating diseases of the liver, and they point out that crude liver extract contains thiamin in considerable quantities.

It is a clinically familiar fact that patients with chronic affections of the liver frequently have anemia in the absence of evidence of blood loss. Wintrobe[64] observed a moderate degree of anemia, mostly macrocytic or normocytic in type, in 37 of 44 cases of cirrhosis, in 32 of 36 cases of primary and metastatic neoplasm of the liver and in 33 of 52 cases of miscellaneous hepatic disorders. He found that the cases of macrocytic anemia responded to intramuscular liver therapy, and concluded that macrocytic anemia results in cases of hepatic disease from an inability to store a necessary hematopoietic principle. Stewart and Rourke[57] found anemia to be the rule in a series of cases of obstructive jaundice, as did Butt, Snell and Keys.[51] Such findings indicate the possible usefulness of liver extract, iron and blood transfusion in the treatment of chronic liver disease, particularly in relation to preoperative and postoperative care.

During the last ten years the riddle of the clotting defect so common and so dangerous in obstructive jaundice has been solved in its practical aspects, although certain questions concerning the

biochemical relations involved are still unanswered. The work of Roderick,[65] Dam,[66, 67] Schönheyder,[68] Almquist and Stokstad,[69] Quick, Stanley-Brown and Bancroft,[70] Warner, Brinkhous and Smith[71] and others has shown that the pathologic bleeding tendency in sweet-clover disease in cattle, in dietary deficiency in chicks and in obstructive jaundice and biliary fistula in dogs and man is based on deficiency of plasma prothrombin. This substance, essential for the rapid formation of an effective clot, is part of the plasma globulin and is formed in the liver. Its formation in sufficient quantity is conditioned in some manner, as yet unknown, by vitamin K, a substance present in various vegetable and animal fats. Hypoprothrombinemia may result from inadequate absorption of fat, as in lack of biliary or pancreatic secretion, from suppression of the synthetic functions of the liver, from too little absorptive intestinal mucosa or too rapid passage of the chyme through the intestine and possibly from insufficient intake of foods containing vitamin K, although the last is somewhat doubtful. Thus hypoprothrombinemia may occur in various disorders of the gastrointestinal tract, though the commonest cause of prothrombin values in the hemorrhagic range is obstructive jaundice.[72]

It has been conclusively shown that prothrombin deficiency can be corrected except in extreme liver failure by the oral administration of crude vitamin K extract and bile salts,[72–74] or by parenteral administration of 2-methyl-1, 4-naphthoquinone and various related chemically pure substances.[75–78] Rapidly acting water-soluble derivatives of 2-methyl-1, 4-naphthoquinone, such as the sodium bisulfite addition-product, are now available, and as with other vitamins a wide margin of safety exists between the effective and the toxic doses. Bile salts do not need to be given when the vitamin K active substance is given subcutaneously or intravenously, and the improvement in prothrombin concentration may last a week after a single injection.[78]

In affections of the liver, as in other diseases in which the administration of vitamins is important, the question of adequate dosage presents itself. It cannot be said that the vitamin requirements in health can be closely defined, and the problem is far more difficult in the variations of disease. So-called "subclinical" vitamin deficiencies are probably prevalent among the supposedly healthy, and do not afford sharp criteria with which the effects of various intake levels can be appraised.[79, 80]

Various methods of study have been devised to give information on individual vitamin needs. The concentration of vitamin A and its carotenoid precursors in the plasma can be determined in an application of the reaction described by Carr and Price[81] and others[82, 83]; or the biophotometric measurement of dark adaptation may be made,[84] although there is some evidence that there may be little correlation between the vitamin A concentration in the plasma and the biophotometer reading.[85] Goodhart and Sinclair[86] studied the concentration of cocarboxylase, the diphosphate ester of thiamin, in the blood by a chemical method and found that it varied directly with the amount of total vitamin B_1 in the blood as shown by a biological method. They[87] concluded that the cocarboxylase determination can be used clinically to demonstrate vitamin B_1 deficiency, with certain rare exceptions, as when the ability of the tissues to phosphorylate thiamin is diminished, or when the red-cell or white-cell count is greatly increased, as in polycythemia vera or myeloid leukemia. Thiamin concentration in the urine may be measured,[88] and evidence has been presented to show that correlation exists between the urinary thiamin values and the state of thiamin nutrition.[89] Ferrebee[90] has proposed fluorometric methods for estimating the urinary excretion of riboflavin, convenient and accurate enough, according to him, for clinical use. Progress has recently been reported[91] in the demonstration of pyridoxin (vitamin B_6) deficiency in patients by urinary excretion studies. Methods for the determination of vitamin C in blood and urine have been widely used clinically, and much valuable information has been obtained.[92] Indirect evidence indicative of vitamin D lack can be obtained by measurements of the concentration of calcium and phosphorus in the plasma and urine, and by skeletal x-ray studies. Vitamin K deficiency may be detected by the determination of plasma-prothrombin concentration by the two-stage method of Warner, Brinkhous and Smith[71] or by the simpler method of Quick.[93]

By means of the clinical application of chemical tests wherever possible not only may vitamin therapy be controlled more effectively in the individual case, but information concerning the vitamin needs in various diseases can be accumulated. Should such laboratory data be lacking in the case at hand, the physician would do well to keep in mind that vitamin deficiencies are often multiple, that there is also need for proper amounts of carbohydrate and protein and that the margin of safety between the effective and toxic dose of vitamins is ample. On such grounds the tendency in treating nutritional deficiency is to give amounts of vitamins that are large compared with the estimated needs in health.[94]

In the care of chronic liver disease, and particularly in forms associated with obstruction to bile flow, the advisability of giving the patient cholic acid derivatives must be considered. The digestion and absorption of fats and fat-soluble substances, such as vitamins A, D, E and K, depend on the presence of proper amounts of bile salts in the intestine.[95, 96] In addition, it has been shown that the interruption of the normal flow of bile into the intestine retards the absorption of hemoglobin-building constituents.[43] Ivy and Berman[97] state that glycogen deposition in the liver may be related to the formation and excretion of bile salts into the intestine. It is to be kept in mind that the amount of cholic acid formed by the damaged liver may be reduced, even though the volume of bile is normal, a finding that has been reported frequently[98] in studies of fistula bile or bile withdrawn from the common duct through a surgical drainage tube. This fact may impair the value of the common clinical practice of refeeding fistula bile, and suggests that the cholic acid content of such bile should first be determined.

To be balanced against the improvement in digestion and absorption that may result from the oral administration of bile salts in liver disease are the possible toxic effects on the liver and kidneys. Cholic acid and its derivatives when administered by mouth or intravenously are rapidly and quantitatively excreted in the bile.[99] After the onset of biliary obstruction, cholic acid appears in the blood and urine. When the acid is administered to an animal with biliary obstruction, much of it can be recovered in the urine and the rest is probably broken down in the liver.[99–101] The intravenous injection of sodium dehydrocholate in animals with a ligated common duct produces in many cases albuminuria and cylindruria, elevation of blood nonprotein nitrogen, reduction in urea clearance and lesions in the renal tubules similar to those of uranium poisoning.[101] Whether such experimental observations have any bearing on the impairment of renal function and bile nephrosis of human obstructive jaundice may be open to question, but the evidence deserves consideration. Various cholic acid derivatives have been extensively used in the nutritional treatment of obstructive jaundice without definite evidence of harm,[61] but it is probably a reasonable practice to give these substances only in small doses. In patients with obstructive jaundice and severe liver damage I prefer to correct prothrombin deficiency by giving vitamin K parenterally and to administer bile salts by mouth only after surgical relief of the obstruction.

Disturbances in water balance and in renal function during the course of hepatic affections require careful clinical consideration. The importance of the liver in buffering sudden changes in the composition of the blood has been pointed out repeatedly.[102–104] Crandall and Roberts[104] found an immediate increase in whole-blood volume after infusion of 0.9 per cent sodium chloride solution in dogs with Eck fistula, the increase being due entirely to plasma. In the normal dog such increase does not occur. Skelton[105] observed a rapid response of the liver and intestines to anhydremia, though the total amount of water in these tissues is far less than that in the muscles and skin. Dogs with Eck fistula show a prompter diuresis after drinking water, and exhibit a lasting increase in volume of water consumed and in urine volume, an effect possibly dependent on interference with the water-storage function of the liver.[106] The same results were found after chloroform poisoning, but not after ligation of the common duct or splanchnicectomy. In acute hepatic injury from arsphenamine poisoning, extensive changes in the electrolyte content of the plasma and urine may occur. The outstanding findings, as reported by Soffer, Dantes and Sobotka,[107] are decreased concentration of plasma chloride and carbon dioxide combining power, increase in inorganic phosphate and in lactic acid and pronounced hemoconcentration; the urine volume increases, and there is increased excretion of lactic acid and total protein and sharp reduction in the excretion of chloride and inorganic phosphate.

In a patient with hepatic insufficiency, rising plasma nonprotein nitrogen values, oliguria and edema are ominous signs, and as Jones and Eaton[108] have pointed out, spontaneous diuresis is evidence of improvement. A complication that ranks high in the fatal cases of obstructive jaundice is anuria, and this is particularly apt to occur in the early postoperative course.[109] In a careful study of renal function in 16 patients with obstructive jaundice, Elsom[110] found signs of renal damage in all, including excessive excretion of casts, epithelial cells and leukocytes. Reduction in urea clearance was present in over half the cases, observations that agree with those of Stewart and Rourke[61] in a similar group of cases. Although no conclusive evidence has been adduced in explanation of the renal lesions so commonly found in obstructive jaundice, the pathological findings are definite and characteristic, and have been likened to the renal lesions in transfusion reaction.[111] In connection with the care of the patient with obstructive jaundice after surgical release of the obstruction, it should not be forgotten that large amounts of water and electrolytes may be lost by external

drainage. As much as 2 liters of fluid containing as much as 15 gm. of sodium chloride may be lost daily in this way.[98]

Such evidence points up the need for controlling parenteral fluid therapy as accurately as possible in handling cases of obstructive jaundice, for lowered hepatic and renal reserve and disturbances in plasma protein are important factors in determining the fluid requirements in these cases.

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CASE RECORDS OF THE MASSACHUSETTS GENERAL HOSPITAL

ANTE-MORTEM AND POST-MORTEM RECORDS AS USED IN WEEKLY CLINICOPATHOLOGICAL EXERCISES

FOUNDED BY RICHARD C. CABOT

TRACY B. MALLORY, M.D., *Editor*

CASE 26521

PRESENTATION OF CASE

A fifty-six-year-old man entered the hospital complaining of pain in his chest.

Seven weeks before admission, after drinking ice water, the patient suddenly developed a pain in his left chest anteriorly that was aggravated by breathing and accompanied by shortness of breath, general malaise and weakness. In addition, a dry cough, which he had had for many years, produced a moderate amount of purulent sputum. These symptoms persisted, though the pain became intermittent, and two weeks before admission he was forced to stop work and remain in bed. The temperature for these two weeks averaged 100.2°F., the cough increased and the sputum though never copious was most marked at night and in the morning. He had lost 7 pounds since the onset of his symptoms and had suffered from moderately severe frontal headaches.

On examination the patient was well-developed and well-nourished but appeared acutely and chronically ill, showed evidence of recent weight loss and was slightly dyspneic. He coughed frequently bringing up small amounts of rusty tenacious sputum. Numerous angiomas were present over the chest and abdomen, and on the right buttock there was a 5-cm. reddened area covered with numerous small pustules. Expansion of the left chest was limited, and in its mid-portion there was dullness to percussion both anteriorly and posteriorly. Over this area were many fine and coarse moist rales with suppression of the breath sounds, which had a slight tubular quality. Occasional rales were heard at the right apex. There was no tracheal shift. Examination of the heart was negative. The liver was firm and nontender and extended three fingerbreadths below the costal margin in the right mid-clavicular line. The fundi showed moderate arteriosclerosis with clear disks and no hemorrhage or exudate. Examination of the nervous system was negative.

The temperature was 101°F., the pulse 95, and the respirations 35.

Examination of the urine was negative. Examination of the blood showed a red-cell count of 5,000,000 with a hemoglobin of 75 per cent, and a white-cell count of 38,000 of which 89 per cent were polymorphonuclears. Blood cultures on two occasions were negative. The nonprotein nitrogen of the blood serum was 16 mg. per 100 cc., the chlorides 94.8 milliequiv. per liter and the carbon dioxide combining power 26.3 milliequiv. Sputum cultures grew *Staphylococcus albus,* alpha hemolytic streptococci, and on one occasion a few colonies of beta hemolytic streptococci. No pneumococci were isolated by culture or the mouse method, and no tubercle bacilli or fungi were found in smears. The tuberculin test was negative in a dilution of 1:40,000 on two occasions.

Fluoroscopic examination of the chest showed movement in both diaphragms. There was a small amount of fluid in the left costophrenic angle posteriorly. The process on the left side involved practically the whole left upper lobe and consisted of areas of diffuse density alternating with small round aerated spaces, some of which had the appearance of small cavities. There was no evidence of respiratory shift of the mediastinum, and the left main bronchus was completely visible. In the right-upper-lung field there were lines of increased density with some mottling, particularly in the infraclavicular area.

An electrocardiographic recording showed normal rhythm with a rate of 100. The PR interval was questionably 0.20 second (marked somatic tremor), and there was low voltage of the QRS complex in Leads 1, 2 and 3.

On the sixth hospital day a bronchoscopic examination was performed. The abnormal findings were limited to the left bronchial tree, where there was considerable reddening of the mucosa of the medial wall of the main bronchus. The left-upper-lobe orifice was not seen, due to angulation. The bronchoscope passed well into the left-lower-lobe bronchus, which showed reddening with a moderate amount of secretion. There was no outcropping, and the terminals were normal. The patient continued to run a spiking temperature up to 104°F., and two weeks after admission developed auricular fibrillation, which was controlled by digitalis. Sulfapyridine was given, but stopped because of distressing vomiting. Sulfathiazole also produced vomiting and, in addition, a nonpruritic maculopapular rash over the forearms, backs of the hands, face and lower abdomen. Both the sulfathiazole and ammonium chloride, which the patient had been receiving for his cough, were discontinued, and the rash disappeared in two days. The former was again instituted two days later in half dosage (3 gm. daily), but the skin eruption reappeared. Three weeks after admission the patient became dis-

orientated. A neurological examination was negative. X-ray study of the chest at this time showed no change in the process. Four weeks after admission bronchoscopic examination was repeated. Considerable secretion was found in the trachea, and several cubic centimeters of thick greyish-white purulent material was aspirated. The right main bronchus showed mucosal injection, and coughing expelled purulent secretion from each bronchial subdivision. There was no localized change in the bronchial mucous membrane. On entering the left main bronchus, the lumen was obstructed just inside the carina by what appeared to be inflammatory swelling. The mucous membrane was engorged and injected, and the bronchoscope could not be introduced more than 0.5 cm. into the bronchus.

At about this time, the patient developed an extensive, partially confluent, papular, erythematous eruption over the entire body except the palms, soles and mucous membranes. The consensus leaned toward a septic origin from pulmonary disease rather than dermatitis medicamentosa. Two months after admission a thoracentesis was performed through the eighth left interspace in the posterior axillary line, and 650 cc. of clear, amber fluid withdrawn. At this point the patient complained of pain, so that the procedure was terminated, although the fluid was still running freely. That evening after a mild attack of dyspnea he coughed up a small amount of bright-red blood and later approximately 150 cc. of blood clot; he then became pulseless and died immediately. During the two months of hospitalization the white-cell count showed a gradual decline from the initial figure of 38,000 to 9900.

Differential Diagnosis

Dr. Frederick T. Lord: May we see the x-rays?

Dr. Aubrey O. Hampton: These films were taken at intervals over a period of not quite one month. In the anteroposterior view it seems as if the whole left lung were diseased, but in the lateral it is apparent that only the left upper lobe is involved. It is honeycombed with cavities but in spite of this is not appreciably reduced in size. The interlobar septum between the upper and lower lobes on the left is straight and in about its normal position. In the films taken a few days later the left side of the diaphragm is higher and there appears to be reduction in the size of the left lung.

In the final film, taken twenty-two days later, a great change is evident. The patient was evidently examined in the upright position. Most of the left chest appears to be occupied by fluid and air with a definite fluid level between them, simulating a hydrothorax or pyopneumothorax. However, the upper margin of the air bubble is at the third rib posteriorly, and it definitely does not reach the apex of the pleural cavity.

Dr. Lord: Is this after tapping?

Dr. Donald S. King: No, before tapping.

Dr. Lord: From the examination of the chest, there is little to be added to the information obtained from the x-ray examination. There were rales at the right apex, and in confirmation the films show definite mottling in the upper part of the right-lung field extending fairly well out to the periphery. There was an enlarged liver and some arteriosclerosis. There was lowering of the nonprotein nitrogen and chlorides in the blood. There were negative tests with tuberculin on two occasions. Of two bronchoscopies, the first was negative and the second, three weeks later, showed obstruction of the left main bronchus.

We should like to know more about certain matters. Was there a history of hemoptysis, or of foul odor to the sputum? It would be desirable to have further information about the pleural fluid, that is, the specific gravity, the cellular formula and the results of cultures, guinea-pig inoculation and search for tumor cells.

The abnormal features of the blood chemistry may be ascribed to the disturbances arising in consequence of the present illness.

The negative tests on two occasions with a 1:40,000 dilution of tuberculin are of doubtful significance. Though the use of highly diluted tuberculin has been advocated as a diagnostic test, its value as a measure of activity of a tuberculous process has not been established, and the use of a high dilution in such a case as this is subject to the disadvantage that patients in an advanced stage of the disease are likely to fail to respond to the test. I do not know the margin of error in the routine reliance on a negative test with a dilution of 1:40,000, but it must be considerable. In Ayman's* investigation of 82 patients with active tuberculosis, only 69 per cent gave positive reactions (10 by 9 mm. or more in size) with 0.1 cc. of a 1:50,000 dilution of old tuberculin.

In the use of tuberculin for diagnosis, it is customary to use stronger solutions. In the screening of school children in Massachusetts with tuberculin, a single test is made with 0.1 cc. of a 1:10,000 dilution of old tuberculin. There is, however, a small margin of error unless those who fail to react to this dose are tested with stronger solutions. When the conditions are more favorable and with the patient under observation in a

*Ayman, D.: The intracutaneous quantitative tuberculin test. *J. A. M. A.* 103:154-157, 1934.

hospital, the first dose, except in cases in which unusual sensitivity is to be expected, may well be 0.01 mg. (0.1 cc. of a 1:10,000 dilution), and if there is no reaction to this amount, 0.1 mg. is used as the second dose, followed by 1.0 mg. if the second test is negative.

To come back to the differential diagnosis, it is evident that the patient had some arteriosclerosis. There was arteriosclerotic heart disease. The enlargement of the liver may have been due to chronic passive congestion or cancer. It is obvious that he had a bronchopulmonary suppurative process with probable cavitation. Whether the cavitation was due to lung destruction or bronchiectasis cannot be determined without the use of Lipiodol, but that substance was not employed in this case.

To return to the effusion, passive congestion as a cause may be excluded. It may have been of inflammatory (tuberculous or nontuberculous) or malignant origin, and the evidence does not permit a decision between these possibilities.

The most interesting, and probably the most important, aspect of the problem is the explanation of the mass in the left primary bronchus. A biopsy of this mass might have established the diagnosis. We have to consider two possibilities. The commonest cause of bronchial obstruction, excluding tenacious secretion, is inflammation. Although the bronchoscopist thought it was of inflammatory origin, it must be admitted that he could not tell by inspection alone. There may be evidence of inflammation at the surface on inspection and even under the microscope, and yet there may be an underlying cancer. It is a question whether carcinoma would grow with sufficient speed to appear within a period of three weeks, the interval between the two biopsies, but the possibility may be granted for a rapidly growing neoplasm. There is also the possibility that the mass is tuberculous, and the x-ray appearance of the right apex suggesting tuberculosis lends some support to this hypothesis. The failure to find tubercle bacilli in the sputum is against it, but we should like to know how many specimens of sputum were examined.

There is another interesting matter in respect to the manner of this patient's death. The immediate cause of death seems to have been hemorrhage into the air passages. He coughed up 150 cc. of blood clot and died immediately, probably from asphyxia.

In respect to diagnoses, we can be certain of bronchopulmonary suppuration with cavitation, probably secondary to bronchial occlusion. So far as the nature of the bronchial mass is concerned, I should regard it as either inflammatory or malignant and confess that I cannot make the decision between these possibilities.

Dr. King: We saw the patient and followed him along in the Thoracic Clinic. We thought at first that he had an oat-cell carcinoma, but we could not prove it. Then we began to think of a primary suppurative process but could not identify the organism. We simply did not have a diagnosis when he died. I am still interested in knowing whether you think that is pyopneumothorax or a large cavity in the left apex.

Dr. Lord: That the gas bubble did not reach the apex suggests the possibility that the fluid was in the lung or encysted in the pleura. Six hundred cubic centimeters of clear fluid would, however, be a large amount to be obtained from the lung unless a cyst of the lung were tapped.

Dr. Lowrey F. Davenport: Perhaps I was negligent in not taking a biopsy specimen at the time of the second bronchoscopy. This man was in wretched condition, and I thought at the time that to do so was unwise.

Dr. Francis T. Hunter: I remember this patient well. I might mention something not emphasized in the printed sheet, that is, the character and amount of the sputum. After they put him on postural drainage, he drained an enormous amount of sputum, probably the most tenacious sticky material I have ever seen, with no odor and no organisms, although at one time we found large numbers of branching mycelium. The sputum had been standing around in a warm room, and the mycelium was never found again; we consequently discounted it.

Clinical Diagnosis

Pulmonary sepsis?
Carcinoma of lung?

Dr. Lord's Diagnoses

Arteriosclerosis.
Arteriosclerotic heart disease.
Bronchopulmonary suppuration (? lung abscess or bronchiectasis).
Bronchial mass (? inflammatory or malignant).
Hemorrhage into the air passages.

Anatomical Diagnoses

Bronchiogenic epidermoid carcinoma, left upper lobe.
Lung abscess (? moniliasis).
Pulmonary hemorrhage.
Chronic fibrous pleuritis.
Encapsulated hydrothorax.
Emphysematous blebs, right upper lobe.
Arteriosclerosis, moderate, coronary and aortic.

Pathological Discussion

Dr. Tracy B. Mallory: At autopsy the left pleural cavity was almost completely obliterated by fibrous adhesions. Between the adhesions were various encapsulated cysts containing straw-colored fluid. It must have been one of these that had been tapped. The left upper lobe was virtually replaced by a multilocular abscess cavity into which several bronchi opened. In the wall of one of the bronchi a small mass was found, and a frozen section was made in which no tumor could be recognized, so that the death certificate was signed as pulmonary abscess. When the regular microscopic sections came through, it was apparent that the little nodule was, after all, an epidermoid carcinoma, not much over a centimeter in diameter; but it was large enough to have obstructed the bronchus, and the remainder of the findings were all secondary to it. The cavity at the time of autopsy was almost completely filled with blood, which also plugged the rest of the bronchial tree, as Dr. Lord suggested. A small mass of inspissated secretion, which was found in the cavity along with the blood clot, was sectioned and found to consist of a practically pure mass of mycelium. I therefore think that yeast infection was probably the cause of the abscess.

A Physician: What about the mottling of the right apex?

Dr. Mallory: There were a number of emphysematous blebs and a few old fibrous scars but no active tuberculous process.

Dr. Wyman Richardson: Was the brain examined?

Dr. Mallory: No.

The liver was large and showed chronic passive congestion. The heart was very slightly enlarged and showed a moderate amount of coronary disease.

Dr. Davenport: What was the relation of the mass to the bronchi? Was it possible to have reached it through the bronchus?

Dr. King: I am quite sure that it could not have been reached.

CASE 26522

Presentation of Case

A seventy-three-year-old engineer entered the hospital for investigation of an unexplained fever.

Because of the patient's physical and mental condition, the history was obtained from his wife, who had been with him constantly. Six weeks before admission the patient complained of general malaise and shortly afterward had a severe chill. Two days later he was lethargic and somnolent, and that night had another severe chill followed by profuse sweating, which soaked the bedclothes. The next day he was completely exhausted, feverish, continually perspiring and had one slight chill. For the next two weeks he ran a constant temperature of 99 to 102°F., and at the end of that time entered another hospital, where physical examination and laboratory investigations were unfruitful; the white-cell count on several occasions was 6000, and the only positive findings were a dry, furry tongue and a constant, spiking temperature. During the next three weeks the patient became confused, complained of soreness in his head and had one momentary attack of dizziness, but his temperature decreased slightly. One week before admission he was given Neoprontosil and shortly afterward became pale and delirious, with tachycardia and a barely perceptible pulse. The drug was withdrawn for two days and then reinstituted, with similar though less severe results. During this week before admission, the patient became lethargic and often delirious, and the nurse stated that he had visual and auditory hallucinations, but answered questions rationally. In addition there was incontinence of urine and, on one occasion, of feces. He had lost 50 pounds since the beginning of his illness.

Several years before admission the patient had had two skin lesions, one situated under the knee, the other near the shoulder. These were slightly reddened, rough, irregular, raised blotches, which healed with an ointment. About two years before admission he complained of a feeling of "sour gas" regurgitating into his esophagus after meals. This complaint was investigated at another hospital, where a gastrointestinal series and barium enema were negative. He was treated successfully by a change in diet.

The patient had had typhoid inoculations on two occasions, the last eight years before admission. He had always lived in Maine, except for short visits to the West Indies. The family history was noncontributory.

On examination the patient was well-developed and well-nourished, flushed, perspiring freely and appeared acutely ill. Numerous 2-mm. tan spots were present over the trunk. Rales were heard at both lung bases. Examination of the heart was negative; the blood pressure was 110 systolic, 66 diastolic. The abdomen was negative to palpation, but the spleen was enlarged to percussion. Examination of the nervous system was negative.

The temperature was 101.5°F., the pulse 104, and respirations 36.

Examination of the urine showed a +++ test for albumin and rare hyaline and granular casts.

Examination of the blood showed a red-cell count of 3,880,000 with a hemoglobin of 11.5 gm. (photoelectric-cell technic), and a white-cell count of 17,600, of which 96 per cent were polymorphonuclears, 2 per cent lymphocytes and 2 per cent monocytes. Blood cultures and a blood Hinton test were negative. The sedimentation rate was 59 mm. in one hour. Agglutination reactions were positive up to dilutions of 1:80 for typhoid bacilli and 1:160 for paratyphoid B bacilli, but were negative in all dilutions for paratyphoid A bacilli. The nonprotein nitrogen of the blood serum was 28 mg. per 100 cc., the blood sugar 188 mg. Examination of the stools was negative, and no pathogens were present.

X-ray examination of the esophagus with barium revealed displacement of the esophagus to the right just above the arch of the aorta and displacement of the entire lower portion to the left, with no evidence of obstruction or involvement of the esophageal wall. There was definite enlargement of the mediastinal lymph nodes, particularly on the right side. An area of mottled density, which was present in the right costophrenic angle, corresponded to the general shape and position of a partially calcified middle lobe. The diaphragm was high on both sides, and the upper portion of the right lung was a little more radiant than the left. There was no mediastinal shift. Films taken in another hospital two weeks before admission showed practically the same findings. X-ray examination of the abdomen showed what appeared to be an enlarged spleen. The liver did not appear to be enlarged, and the visible bones were normal.

The patient developed a brassy cough and signs of respiratory obstruction. His fever continued, ranging between 101 and 103.5°F. He was given sulfanilamide, one 500-cc. transfusion, and two x-ray treatments of 50 r each to the mediastinal mass. Despite these measures the patient died on the sixth hospital day.

Differential Diagnosis

Dr. Lowrey F. Davenport: We have to discuss the case of a seventy-three-year-old man whose outstanding symptom at the time of admission was unexplained fever. The onset with a chill makes us think in the beginning of some acute infectious process starting abruptly. Apparently that was also the thought of those treating him at the other hospital, because blood cultures were taken, and although they were negative, he was treated with Neoprontosil. In a man of seventy-three with this degree of fever, we might reasonably expect the mental confusion described in the record prior to admission here.

What is helpful in the past history? What clues are we given? We are told that he had always lived in Maine, except for visits to the West Indies. That brings up the question of some type of obscure infection that occurs in semitropical regions. He had two negative blood cultures. Agglutination tests were equivocal. We are told that he had been inoculated twice previously with typhoid vaccine and we know that during any infectious process the titer of a Widal agglutination test does go up. The fact that it was weakly positive for both typhoid and paratyphoid B bacilli makes us dismiss typhoid fever as a possibility.

On examination at entry, the patient was obviously ill, in respiratory distress, and running a high fever. Certain additional data on physical examination seem to be significant. They were the enlarged spleen and the numerous pigmented areas over the body. The person taking the history must have been impressed with the pigmentation. He obtained from the wife a story of two earlier skin lesions, one on the leg and one near the shoulder. They appear to have healed quite promptly, and I believe that we can dismiss them.

I am prepared at this point to exclude typhoid fever. We have two positive blood cultures that would tend to exclude, but not necessarily, other acute bacteremic processes. There is nothing in the physical examination to indicate a hidden focus of sepsis to explain this fever. So at this point let us have the benefit of the x-ray evidence.

Dr. George W. Holmes: I presume that these films were taken with the patient lying down. This would account to some extent for the high sharply domed diaphragm. But the diaphragm is rather high even for a patient in the prone position, the right side especially. The mediastinal shadow is certainly increased to the right, and the aorta displaced toward the left. From the character of the mass we should expect that it represented lymph nodes. It is stated in the fluoroscopic note that the esophagus was also displaced, suggesting that the mass was in the region of the peribronchial nodes. In the region of the middle lobe, there is increased density, and when films were taken with the Bucky diaphragm, areas that suggest calcification became apparent. In some of the films there is a suggestion that the upper portion of the right lung is unusually bright as compared with the left side. That would be additional evidence that there was some collapse of the middle lobe with an overdistention of the

upper lobe. Although we have no positive evidence, we do have rather good suggestive evidence of a collapsed middle lobe, with massive enlargement of the peribronchial nodes on the same side.

Dr. Davenport: We still have unexplained fever and an enlarged spleen and now enlarged hilar nodes to tie into the picture if we can. I believe we can say with reasonable certainty that we have excluded all acute infectious processes. There is no reason to believe that it is an infectious granuloma. I think that most of the cases of infectious granuloma are accompanied by a suppurative process, which is particularly true in the lung and invariably in the skin, with the possible exception of the early stages. We are told in the record that the middle lobe was calcified.

Dr. Aubrey O. Hampton: I agree with Dr. Holmes that the more correct interpretation is a partially collapsed middle lobe, with calcification only in the lymph nodes.

Dr. Davenport: That changes the picture. If the middle lobe were partially collapsed and calcified we should assume a long-standing process. If a partial collapse of more recent origin is present, we have to consider something of more recent origin in the bronchus. There is still nothing about any of the data we are given to support the diagnosis of a granuloma. We come down, then, to the question of cancer, a tuberculous process or lymphoblastoma.

Could this be cancer? It would be most unusual for a primary bronchiogenic carcinoma to produce lymph-node involvement of this extent without showing more evidence of lesions in the parenchyma of the lung. We can also say definitely that with primary bronchiogenic cancer the patient should not have had that much fever without more evidence of bronchial obstruction. Is it a metastatic lesion? Again, it is unusual to find metastases in the hilar nodes without metastatic deposits in the lungs, ribs or in the bones of the pelvis.

Could this be tuberculosis? If it were tuberculosis, it would have to be a generalized tuberculosis. We have seen a few cases, even at this age, in which tuberculous nodes have broken down and the organisms have spread through the entire body, causing acute initial symptoms and rapid death. The disease in this patient had been going on for a good many weeks. We have no evidence of a miliary tuberculous process in the lungs to confirm that diagnosis.

I think that we must come down to the diagnosis strongly intimated by the clinical history, namely, lymphoblastoma, probably of the Hodgkin's type. Having reached this diagnosis by exclusion, can we backtrack and make it fit this picture? I think we can. In the past, Dr. Holmes and Dr. Mallory have assured us that it can arise at any age. We know that in certain types of lymphoblastoma, particularly those involving the chest, a high fever occurs. The history of this fever — starting abruptly then subsiding and becoming elevated a second time — is quite characteristic of the type known as Pel-Epstein fever, which is seen in Hodgkin's disease. The moderately enlarged spleen, the evidence of secondary anemia, the white-cell count with high polymorphonuclears all fit the picture. The pigmentation in the skin seems to have been rather nondescript, and widely variable types of skin pigmentation have been reported in cases of lymphoblastoma.

Can we fit all the chest findings into the picture? We are on more shaky ground here, but I judge that the process in the right middle lobe was an old one. The right middle lobe was only partially collapsed. If we want to "go out on a limb," we might guess that this could have been due to previous tuberculosis, primarily bronchial tuberculosis to account for the sharp localization of the process to the middle lobes. We need not necessarily conclude that the two conditions are related, but we should not forget the old dictum that "Hodgkin's disease follows tuberculosis like a shadow." I should make as my diagnosis lymphoblastoma of the Hodgkin's type, with an incidental finding of localized disease in the right middle lobe, probably tuberculosis.

Dr. F. Dennette Adams: I have nothing to add except to say that Dr. Davenport's reasoning was very similar to ours when we had the patient in the hospital. One thing that baffled us was that four or five days before he died the white-cell count, which had previously been low, suddenly became high. Dr. Chester Jones considered that a strong indication that we were dealing with some sort of acute infection, just what, he did not know; neither did I.

Clinical Diagnosis

Lymphoma.

Dr. Davenport's Diagnoses

Lymphoma, Hodgkin's type.
Localized disease of the right middle lobe (? tuberculosis).

Anatomical Diagnoses

Tuberculous lymphadenitis of paratracheal and bronchial nodes.
Tuberculous ulcer of the esophagus.

Miliary tuberculosis.
Hypertrophy of the heart.
Arteriosclerosis, moderate: aortic and coronary.
Hyperplasia of the prostate.

Pathological Discussion

Dr. Tracy B. Mallory: At autopsy we were somewhat surprised by the findings. The most obvious one was a large ulcerated mass in the esophagus, with raised edges and a deeply ulcerated, perforated base. The bronchial and peribronchial nodes were normal. The pericardium was studded with miliary lesions, and the prosector was perfectly satisfied that it was a case of carcinoma of the esophagus with extensive metastases. However, that was not the end of the story, because when sections were examined it was evident that this ulcer of the esophagus was caseous and contained enormous numbers of tubercle bacilli. We then had to reconstruct our ideas. It quickly became evident that the primary lesion was a very extensive tuberculosis of the bronchial lymph nodes, secondarily ulcerating into the esophagus and producing as the terminal event a generalized miliary tuberculosis. The enlargement of the spleen was due to multiple miliary tubercles, which were also present in the liver and were quite numerous in the lungs, despite the x-ray films. There was no tumor anywhere. We were not impressed with any collapse of the middle lobe.

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THE MEDICAL CARE OF THE SICK IN THEIR HOMES

IN 1795, Boston, within whose "harbour there is room enough for near 500 sail to lie at anchor, and in good water too," boasted of some 18,000 inhabitants, and within that year Dr. John Fleet, first district physician to the Boston Dispensary visited 80 patients in their homes—patients too ill to leave their beds, yet too poor to be able to afford a private physician. Since then the work of the Boston Dispensary, established to "provide medical advice and relief to the sick poor," has increased and multiplied and prospered until in 1939 its "family physicians" made 54,500 house visits on over 30,000 patients.*

Many of Boston's leading physicians of the older generation look back on their district work as one of the most valuable experiences of their early professional careers. Many a Tufts medical student of today has, under the aegis of the New England Medical Center, in a similar role gained valuable bedside experience, and under the guidance of a district physician has given timely care to those whose need was great.

Thus Boston, unlike other cities of the country, has profited by the services of a privately supported medical charity. Through the years, however, it became increasingly necessary to appeal to the city government for financial aid, and with the probable dwindling of voluntary philanthropic contributions it is indeed questionable whether such a service can be supported, even in part, by private charity.

If the Boston Dispensary and other agencies concerned are to be relieved by the City of Boston of the financial burden of domiciliary medical care, the question remains as to how best the work may be carried on. Many suggestions have been made, but there is a preponderance of opinion that, for Boston, the ideal arrangement would be through a contractual agreement between the city and the Boston Dispensary whereby the poor might be assured of service of the high level that has been maintained for many years.

Whatever the eventual outcome may be, it is apparent that the problem of domiciliary medical care of the indigent sick is of great importance, and that it deserves the careful consideration of all concerned.

*Wing, F. E.: *Medical Care of the Sick in Their Homes*. 31 pp. Boston: The Boston Dispensary, 1940.

SKI PATROLS ON TRAILS AND SLOPES

THIS winter, between three and five million people are expected to participate in skiing. With the tremendous growth in the popularity of the sport, many serious and minor accidents have occurred on the trails, principally because so many novices were attempting feats beyond their skill and endurance. The number of injuries has been reduced by the efforts of the voluntary patrols maintained by almost every ski club in the country. The American Red Cross has helped standardize the first-aid methods, rescue practice and trans-

portation equipment, the principles of which are contained in its booklet, *Ski Safety and First Aid*.

During the coming season, the members of the patrols plan to emphasize the prevention of accidents, but, as in previous years, they will also be on the alert to help the injured. On many popular trails the Red Cross and some ski clubs have established caches in which blankets, toboggans, heating pads and other necessary equipment are kept ready for emergencies.

First-aid procedure involves considerable skill and diligence: the loaded toboggan must be maneuvered slowly and carefully down the trail, so that the injured person may not be in danger of further hurt; night may come before an ambulance reaches the sufferer; blankets and splints must be brought back from the hospital; equipment must be returned to the cache for further need; and dressings and bandages must be replaced.

The work of preventing accidents and administering first aid is done in the holiday time of the patrol members, who forego their own recreational skiing and make long, tedious and often dangerous trips to assist skiers in distress. In many sections the patrols cover all slopes and trails at the end of the day to be sure that nobody has been left on a mountain with an injury or broken equipment.

To promote safety in skiing, the patrols direct beginners to practice slopes, where they may receive instruction before attempting the more difficult and dangerous trails. Both novices and experienced skiers are cautioned not to start down a steep slope unless they know how to slow down and stop. By wearing goggles while climbing, the skier may avoid extremely painful and often serious snow blindness. Beginners are instructed to stay close to the side of the trail when climbing, to slow down when rounding blind turns and to travel in groups and remain together. They are also told to observe snow conditions and are warned that the cold weather aggravates injuries that under different circumstances might be slight.

This increased interest in skiing is a sign of progress — so far as the general health of the people is concerned — in an age when the use of automobiles has largely eliminated walking as an exercise; furthermore, these efforts of the American Red Cross and the ski clubs to prevent accidents and to render adequate first-aid treatment deserve proper recognition.

MEDICAL EPONYM

Tetralogy of Fallot

This syndrome was described by Arthur Fallot in a series of papers, entitled "Contribution à l'anatomie pathologique de la maladie bleue (cyanose cardiaque) [Contribution to the Pathologic Anatomy of the Blue Sickness (Cardiac Cyanosis)]," which appeared in *Marseille-Médical* (**25:** 77–93, 138–158, 207–223, 270–286, 341–354 and 403–420, 1888.

A portion of the translation (page 419) follows:

> The blue sickness, especially when diagnosed in the adult, is the result of a small number of perfectly definite cardiac malformations.
>
> Of these cardiac malformations, one exceeds all the others in frequency, since we have found it in nearly 74 per cent of our cases. . . .
>
> This malformation is a true anatomicopathological entity, represented by the following tetralogy: stenosis of the pulmonary artery, communication between the ventricles, displacement to the right of the origin of the aorta, and hypertrophy of the right ventricle, almost always concentric in type. To these may occasionally be added, with only accessory significance, a persistent ductus Botalli.

R.W.B.

MASSACHUSETTS MEDICAL SOCIETY

RESOLUTION ON THE DEATH OF DR. EDWARD J. DAILEY

Whereas, An old wise Father has seen fit to call from among our midst our friend and colleague, Dr. Edward J. Dailey, and

Whereas, This tragic death, which makes us pause and reflect on the uncertainties of life, brings a realization of how great a loss the profession has sustained by his untimely death; be it therefore

Resolved, That in the death of Dr. Edward J. Dailey, a long, active and useful career has been brought to a close. We mourn his loss as a brother physician, as a loyal citizen and as a genial friend. And be it further

Resolved, That these resolutions be published in the *New England Journal of Medicine* and the *Somerville Press*, placed on records of the society,

and a copy sent to the bereaved family with our deepest sympathy.

RESOLUTION COMMITTEE
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RESOLUTION ON THE DEATH OF DR. HENRY JACKSON

The following resolution was presented by Dr. Paul D. White at the meeting of the New England Heart Association, held at the Massachusetts General Hospital, Boston, on December 16, 1940, and was unanimously adopted:

INASMUCH AS, The New England Heart Association has recently lost by death its first president, Dr. Henry Jackson, who served from 1921 to 1926,

INASMUCH AS, He was for many years a leader in the study of heart disease and an inspiring and devoted member of our association, and

INASMUCH AS, We shall greatly miss his wise counsels and friendly spirit in our midst, be it

RESOLVED, That we hereby record our gratitude for his service in this field and send to his family this resolution with our deepest sympathy.

EDWARD F. BLAND, M.D., *Secretary*.

SECTION OF OBSTETRICS AND GYNECOLOGY*

RAYMOND S. TITUS, M.D., *Secretary*
330 Dartmouth Street
Boston

DIABETES MELLITUS AND PREGNANCY

Since October 10 the weekly cases in the *Journal* have concerned diabetes mellitus as a complication of pregnancy. It is evident from these that the diabetic mother presents no particular obstetric problem. Insulin has made it perfectly safe for such mothers to become pregnant provided that during pregnancy they are treated intelligently from the diabetic standpoint.

The greatest problem in diabetes in pregnancy is fetal mortality. Any study of diabetic pregnancies brings out the frequency with which hydramnios is noted as a complication, and in diabetic patients this hydramnios usually exists without the association of twins or fetal abnormalities — conditions associated with hydramnios in the pregnant nondiabetic patient. The treatment of this hydramnios is, of course, an obstetric problem. Toxemias are much commoner in diabetic than in nondiabetic mothers, and acute toxemias present the same obstetric problem in the diabetic that they do in the nondiabetic pregnant woman. In these ways only do diabetic mothers present particular problems.

Insulin therapy has not prevented spontaneous abortions, miscarriages, stillbirths and neonatal deaths. These occur, particularly stillbirths, in a much greater proportion in diabetic than in nondiabetic women.

The frequency of the incidence of toxemia led Smith and Smith to inaugurate prolan, estrin and progestin metabolism studies on patients with diabetes. Some authorities believe that these studies should be routine on all diabetic women who are pregnant. Substitutional therapy has resulted in the control of these toxemias in many cases, and a rise in the fetal survival from 42 to 88 per cent. The replacement therapy consists of the intramuscular injection of from 150,000 to 300,000 international units of Progynon B and 10 to 40 mg. of Proluton daily. Since the cost of this therapy is almost prohibitive, the oral administration of stilbesterol and Pranone has been recommended. The latter are not so efficient as those given intramuscularly. In a small series of patients intramuscular stilbesterol appears to be effective. Too few cases have been treated to be dogmatic about the use of this therapy, but the results have been highly gratifying.

Cesarean section in the treatment of diabetic patients offers no argument when the section is done because of acute toxemia or because of a fetus that is too large for the individual pelvis. For the purpose of ensuring fetal survival, it is still advocated in mothers with severe diabetes contracted in childhood, when their pregnancies have reached thirty-seven weeks or more.

The next subject to be discussed will be heart disease in pregnancy.

*A series of selected case histories by members of the section will be published weekly. Comments and questions by subscribers are solicited and will be discussed by members of the section.

DEATHS

CHIPPENDALE — FRANCIS DAVID J. CHIPPENDALE, M.D., of Fall River, died November 19. He was in his twenty-ninth year.

Born in Fall River, he attended the University of Maryland, Canisius College, and received his degree from the Georgetown University School of Medicine in 1936.

He was a member of the Massachusetts Medical Society, the American Medical Association and the Fall River Medical Society.

His mother, five brothers and two sisters survive him.

DAILEY — EDWARD J. DAILEY, M.D., of Somerville, died December 10. He was in his sixty-second year.

Born in Cambridge, he received his degree from Tufts College Medical School in 1904. In 1920 he founded the

Somerville Central Hospital and at the time of his death was the superintendent there.

Dr. Dailey was a fellow of the Massachusetts Medical Society and the American Medical Association, and held memberships in the New England Hospital Association and on the American Board of Hospital Directors.

His widow, three sons, three daughters, a sister and a brother survive him.

HEWITT — CLARENCE E. HEWITT, M.D., of Springfield, died December 18. He was in his seventy-ninth year.

Born at Lebanon, New York, he attended Cazenovia Seminary, New York, and received his degree from Baltimore Medical College in 1897.

Dr. Hewitt had practiced medicine in Springfield for forty-three years and was a fellow of the Massachusetts Medical Society and the American Medical Association. He was also a member of the Springfield Academy of Medicine.

His widow, a sister and a brother survive him.

NEW HAMPSHIRE MEDICAL SOCIETY

DEATH

BURTT — EDWARD D. BURTT, M.D., died at his home in Lincoln on December 2. He was born in Plymouth, New Hampshire, on November 21, 1871, the son of Frederick and Leonora (Wheeler) Burtt. He studied medicine at Vermont University and graduated from the Baltimore Medical College in 1900.

Dr. Burtt was a member of the New Hampshire Surgical Club, a member of the American Medical Association and a member of the New Hampshire Medical Society.

He is survived by his widow, one daughter, four brothers and two sisters.

HENRY H. AMSDEN, *Necrologist,*
New Hampshire Medical Society.

VERMONT STATE MEDICAL SOCIETY

VERMONT NEWS

National Red Cross headquarters in Washington, D. C., will offer women of Burlington and vicinity, including women who are students at the University of Vermont, the opportunity of learning how to be instructors for courses in first aid and life saving. These two subjects will be taught in an eighteen-day course, with Mr. N. A. Parker of the Washington staff of the Red Cross as instructor, at the Burlington Community Y. M. C. A., March 3 to 21, 1941. The lessons will be given on alternate nights throughout the course. Satisfactorily completing the courses will mean receiving certificates carrying the right to teach first aid and life saving.

Those who wish to take the life-saving course in order to qualify as life-saving instructors must have earned their rating as senior life savers. Another requirement for those taking the life-saving course is fifteen hours of preliminary training, which enables Northerners, who lack all-year-long swimming opportunities, to brush up on the fine points of swimming. This fifteen-hour preparatory course will be given this winter at the Community Y. M. C. A.

MISCELLANY

NEW DEAN FOR BOSTON UNIVERSITY SCHOOL OF MEDICINE

The selection of Dr. Bennett F. Avery of Beirut, Syria, a well-known anatomist, as dean of Boston University School of Medicine has been recently announced. He will succeed the late Alexander S. Begg and will be the fourth dean to head the school since its adoption by Boston University in 1873. Being thirty-nine, Dr. Avery is one of the youngest men ever to become dean of a United States school of medicine. He received B.S., M.S. and M.D. degrees from the University of Michigan, where he taught anatomy for one year under Dr. G. Carl Huber, and in 1926 went as adjunct professor of anatomy to the American University in Beirut. He was later advanced to the rank of full professor and was also made acting dean of the school. At present he is in this country on sabbatical leave as visiting professor of anatomy at Yale University School of Medicine. He will assume his duties at Boston University on January 1. Dr. Avery has returned periodically to this country to do research work at the universities of Michigan, Chicago and Yale.

CORRESPONDENCE

LOVERING FAMILY

To the Editor: We are compiling a family tree of the Lovering family that originally sprang, many years ago, from David Lovering, of Greenwood, Maine. He carried the hereditary Charcot-Marie-Tooth type of progressive muscular atrophy, involving the hands and feet. His descendants have also gone under the names of Woodis, Stone, Rowe, Francis and Wardwell.

Any physician who has had cases of this disease, especially in patients of the above names, is kindly requested to get in touch with us.

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REPORT OF MEETING

HARVARD MEDICAL SOCIETY

A regular meeting of the Harvard Medical Society was held at the Peter Bent Brigham Hospital on October 8, with Dr. Elliott C. Cutler presiding.

The first case, presented by the medical service, was that of a young man with a demonstrable duodenal ulcer who had had recurrent episodes of bleeding associated with anemia, fatigue and, finally, a fainting attack that brought him to the hospital. Dr. Eugene A. Stead, in discussing the case, emphasized that this could indeed be considered a type of "shock" despite the lack of physical findings that are usually considered characteristic of this condition. In regard to the rationale of blood transfusions in such cases, Dr. Stead pointed out that although the introduction of additional fluid does not ordinarily raise the blood pressure in the normal person, it is a well-established surgical fact that there is more bleeding from such lesions as ulcers if blood is introduced during a hemorrhage. He suggested in general, therefore, that blood transfusions in

hemorrhage be withheld from young patients in good condition until the clot has become fairly firm, and that during recovery such therapy be carried out freely. Dr. Cutler approved of this regimen and added that few people die of hemorrhage from natural causes if left alone.

The second case, presented by the surgical staff, was that of an elderly woman who entered in an aphasic state with incomplete history of abdominal pain of forty-eight hours' duration. There had been no bowel movements; only fluids could be tolerated by mouth. The past history was relevant in that an adenocarcinoma of the uterine fundus was diagnosed in 1923, and was treated first by roentgen rays and by panhysterectomy in 1926, when residual disease was still found on diagnostic dilatation and curettage. Physical examination revealed that the patient was slightly dehydrated, with slight right-lower-quadrant tenderness, occasional audible peristalsis, a questionable prepubic mass, and some tenderness but no mass on rectal examination. The blood pressure was 170 systolic, 90 diastolic, and there was a slight apical systolic murmur. The significant laboratory data were a leukocyte count of 17,700 with 84 per cent neutrophils, an erythrocyte count of 5,500,000, a hemoglobin content of 90 per cent and a very slight trace of albumin in the urine. After parenteral fluids were started, flat plates of the abdomen were taken, and the patient was removed to the operating room, where an exploratory laparotomy was performed. The peritoneal cavity contained 250 cc. of blood-tinged fluid, and there were distended loops of reddish-blue ileum. Adhesive bands were severed, and the patient was returned to the ward on Wangensteen drainage. The following day the white-cell count was 15,500, and the red-cell count 4,500,000 with 80 per cent hemoglobin. Blood chlorides were 106 milliequiv., and plasma proteins 6 gm. per 100 cc. The case was presented to illustrate another form of shock and its treatment.

The speaker of the evening was Dr. John Scudder, of Columbia University, whose subject was "Shock: Blood studies as a guide to therapy." In his introduction, Dr. Scudder pointed out that these studies considered shock from the aspect of individual cells and their metabolism. Original studies by Dr. Osterhout on certain unicellular plant cells demonstrated that a concentration gradient exists between the intracellular and extracellular components. Trauma of any sort was shown to cause a breakdown of the barrier, with an escape of the predominant intracellular ion, potassium, and a transfer of sodium in the opposite direction. In Nitella, increasing the concentration of potassium in pond water is measurably changed in the action current.

In mammals, such as the cat, by merely changing the concentration of potassium of the blood, one is able to bring about various changes, visible by the electrocardiograph, that lead to ventricular fibrillation, asystole and death. The administration of sodium salt, by causing a diuresis of the potassium, will bring these electrocardiographic changes back to normal, provided therapy has been instituted early.

That the same fundamental phenomena occur in human shock was demonstrated by the increase of potassium following a ruptured kidney and in untreated burns. Further observations revealed that there was a measurable increase of the specific gravity of the peripheral blood, although the hematocrit reading and other routine laboratory data remained unchanged. Dr. Scudder urged that the therapy of shock cases be based, therefore, on the hematocrit reading, the specific gravity of the whole blood and plasma, and the plasma protein, rather than on changes of blood pressure or ionic concentration, which occur only later.

The speaker then discussed the background and the methods employed at his clinic for carrying out these determinations easily and practically. The need for performing all four tests, rather than any single one, was emphasized, and this was illustrated by cases of shock wherein the hematocrit reading or the plasma protein, or both, remained well within normal limits despite a dangerous degree of shock. Dr. Scudder pointed out that an adequate level of protein is an aid against dangerous shock. Decrease in plasma proteins is usually found in severe cases of burns, combined with great hemoconcentration. This is a situation difficult to combat and should be treated by the injection of plasma rather than of whole blood.

The speaker demonstrated the ingenious apparatus whereby the specific gravity of whole blood and plasma may be simply and accurately determined by an application of Stokes' law of falling bodies. A measured drop of the appropriate fluid is put in a tube and the speed of its descent timed with a stopwatch and compared with that of a fluid of known specific gravity. Then the utilization of the linear relation between the plasma protein content and the plasma specific gravity, originally suggested by van Slyke, allows the former value to be computed readily.

Dr. Scudder asserted that an intelligent appraisal of these four determinations results in an accurate and rapid appreciation of the underlying pathologic physiology in cases of shock and thus leads to the early adoption of the appropriate line of therapy to be employed. The speaker himself was first interested in these studies following his experience with Asiatic cholera epidemics in India. The mortality drop from 80 to 20 per cent during the last century has resulted through intelligent use of proper fluids.

Physiologic saline solution is often of advantage early in cases of shock to correct dehydration and the ionic equilibrium before blood transfusion is employed. Dr. Scudder again reminded the group of the great lowering of the plasma protein in burns as contrasted with hemorrhage and of the great advantages of using infusions of plasma instead of whole blood in the former condition.

He pointed out that some patients who fail to respond properly to intravenous saline may continue to have increasing hemoconcentration. The use of adrenocortical extract in such cases has proved to be a corrective measure in both animals and man. It was suggested that there was a premature breakdown of the sympathicoadrenal mechanism in these rare cases.

Finally Dr. Scudder briefly discussed the problems involved in preserving and using reserve blood. Studies have revealed that there is a gradual loss of leukocytes, particularly polymorphonuclear neutrophils, whereas the erythrocytes and the hemoglobin content remain essentially unchanged. A small loss of cell volume is associated with a diminution of potassium. It was therefore suggested that as small an interface as possible be employed in storage flasks to decrease the possibility of ionic loss. Withdrawal of blood under carbon dioxide was found beneficial, and sodium citrate was thought the best anticoagulant.

Plasma is drawn off after one week and has been found beneficial in practical experience. As was pointed out, this was to be expected as the result of electrophoretic charts, wherein plasma more nearly approaches blood than any other fluid. Lyophilic serum was shown to fall down in this respect, and it was said that its use has not proved practical. Plasma, mixed with equal parts of saline, has proved very beneficial in surgical emergencies.

The discussion was opened by Dr. S. J. G. Nowak, of the

Boston City Hospital, who reported on cross-perfusion experiments that appeared to show that there is no circulating breakdown substance responsible for shock. Dr. Stead remarked that medical shock may often be observed when there is no disproportion between the existing vascular bed and the circulating blood volume. Therefore there may be no change of any of the factors used as routine criteria of shock. On the other hand, he has been impressed by the amount of change in the blood constituents that may occur without shock. He cited cases with plasma protein levels that rose from 6.8 to 8.0 gm. per 100 cc.

Dr. J. E. Dunphy stated that the characteristic clinical picture of shock usually appears when treatment is no longer of avail. Therapy must be instituted early, particularly in cases of peritonitis, intestinal obstruction and so forth, which are now seen more commonly than those due to trauma or burns. And it is particularly here that Dr. Scudder's procedures allow accurate fluid therapy. Dr. Dunphy has been impressed, however, with the need for clinical as well as laboratory judgment, for example, when the protein value may appear normal initially but may need supporting when hemoconcentration has been overcome.

In conclusion, Dr. Scudder introduced the problem of why adrenocortical extract sometimes exerts a deleterious effect, particularly in peritonitis. He suggested as a possible mechanism the action of this extract in suppressing the potassium content, as shown by Loeb. Desoxycorticosterone may cause a fall from 5.0 to 2.5 milliequiv., with cardiac standstill. And this effect is even more serious in peritonitis, in which the potassium value often reaches levels of 3 milliequiv.

NOTICES

BOSTON DOCTORS' SYMPHONY ORCHESTRA

The Boston Doctors' Symphony Orchestra will rehearse under Alexander Thiede, formerly concertmaster of the Cleveland Symphony Orchestra, every Thursday at 8:30 p.m. Those interested in becoming members should communicate with Dr. Julius Loman, Pelham Hall Hotel, Brookline (BEA 2430).

NEW ENGLAND HOSPITAL FOR WOMEN AND CHILDREN

The monthly clinical conference and meeting of the staff of the New England Hospital for Women and Children will be held in the classroom of the Nurses' Residence on Thursday evening, January 2, at 7:15 p.m. Dr. Merry Pittman will be chairman.

PROGRAM

Electrocardiographic and Roentgenologic Evidence of Heart Disease with Demonstration of the Stethocardiette. Drs. Merry Pittman and Bianca R. Lia.

MASSACHUSETTS DEPARTMENT OF CIVIL SERVICE AND REGISTRATION

Medical Inspector of Schools, Health Department, $836.57 a Year, Fall River

Director of State Civil Service, Ulysses J. Lupien, has announced that competitive examinations are to be held on January 18 to find eligibles for appointment to the position of medical inspector of schools, Health Department, Fall River.

The entrance requirement is as follows: applicants must be registered physicians under the Massachusetts Board of Registration in Medicine. The subjects and weights of the examination are as follows: training and experience, 2; practical questions, 3; total, 5. Applicants must obtain a grade of 70 per cent in each subject in order to become eligible. The last date for filing applications is January 4, at 12:00 noon.

SOCIETY MEETINGS AND CONFERENCES

CALENDAR OF BOSTON DISTRICT FOR THE WEEK BEGINNING SUNDAY, DECEMBER 29

MONDAY, DECEMBER 30

12:15–1:15 p.m. Clinicopathological conference. Peter Bent Brigham Hospital amphitheater.

TUESDAY, DECEMBER 31

12:15–1:15 p.m. Clinicoroentgenological conference. Peter Bent Brigham Hospital amphitheater.

DECEMBER 27 — Waltham Medical Meeting. Page 1039, issue of December 19.

DECEMBER 27 — Danvers State Hospital, clinicopathological conference. Page 999, issue of December 12.

DECEMBER 27–29 — National Convention of the Association of Medical Students, Boston.

JANUARY 2 — New England Hospital for Women and Children. Notice above.

JANUARY 3 — Massachusetts Memorial Hospitals, staff meeting. Page 999, issue of December 12.

JANUARY 4 — American Board of Obstetrics and Gynecology. Page 787, issue of November 7.

JANUARY 5–MARCH 2 — Public Health Lectures. Cambridge Hospital. Page 1039, issue of December 19.

JANUARY 7 — Greater Boston Medical Society. Page 1039, issue of December 19.

JANUARY 9 — Pentucket Association of Physicians. Page 263, issue of August 15.

JANUARY 13, 14 — Third Annual Congress on Industrial Health. Page 999, issue of December 12.

FEBRUARY 20–22 — American Orthopsychiatric Association, Inc. Page 999, issue of December 12.

MARCH 8 — American Board of Ophthalmology. Page 201, issue of August 1.

MARCH 12–14 — New England Hospital Assembly. Hotel Statler, Boston.

APRIL 21–25 — American College of Physicians. Page 1065, issue of June 20.

MAY 21, 22 — Massachusetts Medical Society, Boston.

JUNE 2–6 — American Medical Association. Cleveland, Ohio.

DISTRICT MEDICAL SOCIETIES

ESSEX SOUTH

JANUARY 8 — Visceral Pain: Its relief. Dr. James C. White. Danvers State Hospital, Hathorne.

FEBRUARY 5 — Gastric and Duodenal Ulcer: Diagnosis and treatment. Dr. Arthur Allen. Lynn Hospital.

MARCH 5 — X-ray in Heart Disease. Dr. Merrill C. Sosman. Essex Sanatorium, Middleton.

APRIL 2 — Pediatric Problems in General Practice. Dr. Joseph Garland. Addison Gilbert Hospital, Gloucester.

MAY 14 — Relation of the Doctor to the Law. Mr. Leland Powers. New Ocean House, Swampscott.

FRANKLIN

JANUARY 14.

MARCH 11.

MAY 13.

Meetings will be held at 11 a.m. at the Franklin County Hospital, Greenfield.

NORFOLK

JANUARY 28 — Carney Hospital.

FEBRUARY 25 — Medicolegal meeting. 8:30 p.m. Hotel Puritan, Boston.

MARCH 25 — To be announced.

MAY 8 — Censors' meeting. Hotel Puritan.

SUFFOLK

JANUARY 29 — Page 604, issue of October 10.
APRIL 30 — Page 604, issue of October 10.

WORCESTER

JANUARY 8 — Worcester City Hospital, Worcester.
FEBRUARY 12 — Worcester State Hospital, Worcester.
MARCH 12 — Memorial Hospital, Worcester.
APRIL 9 — Hahnemann Hospital, Worcester.
Supper at 6:30 p.m. followed by a business meeting and scientific program.

BOOK REVIEWS

Dermatologic Therapy in General Practice. By Marion B. Sulzberger, M.D., and Jack Wolf, M.D. 8°, cloth, 680 pp., with 65 illustrations and 25 tables. Chicago: Year Book Publishers, Inc. $4.50.

This work is the answer to the long-existing need for an intelligible, complete and concise compendium of procedures to be followed in the majority of skin diseases that are encountered by physicians in general practice. The dermatologist can also gain some profit from almost every page in the book. Although practically every procedure described and every preparation and prescription discussed can be found in the general literature, to collect them in a handy form would require an extensive search of the dermatological texts and journals. The work suffers somewhat, however, from an inconsistent mixture of unofficial abbreviations of Latin and English, all mixed together in the same prescription — a fault that occurs quite frequently in many textbooks. Since most prescriptions are written in Latin with proper endings, they all should have been written in the same manner. However, the very extensive formulary and the minutiae of nursing and of the technic of treatment, as given in this book, leave very little to be desired.

The first two chapters are the essence of the book, so far as the general practitioner is concerned; they contain a thorough discussion of the principles of external medication. In the chapter on urticaria, there are an excellent condensation of diet tables and a general description of allergic conditions of the skin. There are over fifty pages of description and detailed instruction for the diagnosis and treatment of fungus infections. The various charts and tables and the outline form of presentation, as well as the pertinent photographs, are valuable features of the book. Even the busiest general practitioner will greatly benefit by reading this very admirable contribution to the practical management of dermatoses.

The Diagnosis and Treatment of Diseases of the Peripheral Arteries. By Saul S. Samuels, A.M., M.D. Second edition. 8°, cloth, 372 pp., with 106 illustrations. London: Oxford University Press, 1940. $6.50.

In this second edition, Dr. Samuels attempts to bring the subject of peripheral arterial disease up to date. The book considers mainly thromboangiitis obliterans and arteriosclerosis. In the former disease, the author still relies largely on treatment with intravenous hypertonic salt solution, and he is content to wait several weeks or months for spontaneous separation of gangrenous parts. All physicians will not agree with his stated policy of never amputating in this disorder. More physicians will, however, be in accord with his emphasis of general hygiene and abstention from smoking.

Although the book presumes to be comprehensive, the sections on disorders other than Buerger's disease and arteriosclerosis are too brief and sketchy to be of much value. The chapter on Raynaud's disease is particularly disappointing, since diagnostic and operative procedures are but briefly mentioned.

Some of the subject matter in the book is actually misleading. Uroselectan is mentioned as a painless material for arteriography; in the reviewer's experience, this is one of the most irritating solutions available. The author's advocacy of hot baths and diathermy in organic arterial disease may be justified by his experience, but he makes no mention of recent observations on the rapid disintegration of ischemic tissue in the presence of a heated environment.

It seems advisable to sound a note of caution and to specify what patients should be treated in this manner. The emphasis on instrumental measurement rather than palpation of the pulses seems most unfortunate. Thus on page 31, the author states, "palpation of the posterior tibial artery is notoriously unreliable," and again on page 28, "in a crude way, digital palpation of peripheral arteries may give some clue concerning the condition of the circulation." Finally, the tone of the book is much too dogmatic for a subject that to a great extent is still controversial. The book seems to extend but little our knowledge of vascular disorders.

Adventures of a Biologist. By J. B. S. Haldane, M.A. Second edition. 8°, cloth, 281 pp. New York and London: Harper & Brothers, 1940. $2.75.

Professor Haldane, a distinguished scientist, a materialist and a Marxist, takes the reader behind the scenes to let him know what sort of questions interest the scientist. He draws attention not only to the unsolved but also to the unasked problems. His essays, by-products of scientific teaching and research, cover a broad field ranging from discussions on weather, earth and sun to life, race, health, death, philosophy and politics. The point is emphasized throughout that man is a machine but at the same time an individual, so that life becomes an extraordinary bundle of contradictions. A warning is given against harnessing biology to the car of any political party. On the other hand the author is strongly of the opinion that scientists should take an active part in politics, since the questions asked in the biological sphere will depend on our economic and political orientation. This is a book that may be recommended without reservations.

A Text-book of Psychiatry for Students and Practitioners. By D. K. Henderson, M.D. (Edin.), F.R.F.P.S. (Glas.), F.R.C.P.E., and R. D. Gillespie, M.D. (Glas.), F.R.C.P. (Lond.), D.P.M. (Lond.). Fifth edition. 8°, cloth, 660 pp. London: Oxford University Press, 1940. $6.00.

This edition of a standard and good textbook of psychiatry, designed for the use of students and practitioners, is clearly written and fairly comprehensive. It has an important, new chapter on the psychiatry of childhood. There is also a relevant and clear outline of the relation of psychiatry to law. It is not doctrinaire, for it neither rejects nor accepts the various schools which have elaborated theories, especially concerning the functional mental states. There is a good section on the use of insulin and Metrazol, although, unfortunately, the use of convulsive and shock therapy for the depressive and involutional states, where the greatest benefit from these methods seems indicated by present-day experience, is not discussed.

In the present unsatisfactory state of psychiatry, so far as classification, pathology and treatment are concerned, this book is as good as any for the beginner in this difficult field.

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